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SPONDYLITIS ANKYLOPOIETICA.

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THE term "*spondylitis ankylopoetica*" has as synonyms "ankylosing spondylitis", "Marie spondylitis", "Strümpell spondylitis", "von Bechterew spondylitis", "*spondylitis deformans*", "rheumatoid spondylitis" *et cetera*. The name "spondylitis" should include any pathological lesion which involves the spinal vertebræ, and therefore tuberculous caries, typhoid spine, senile kyphosis and brucellosis of the spine are within its ambit. However, there are two great varieties commonly seen in practice to which the term spondylitis is applied. The one variety, *spondylitis ankylopoietica*, is generally seen in young people, and therefore has been designated by Gilbert Scott "adolescent spondylitis". It is not, however, confined to the young, but may begin in the later decades of life, producing the same pathological picture of ankylosis of the sacro-iliac joints, the apophyseal joints and the ligaments of the whole spine. The other variety, *spondylitis osteoarthritica*, is common in older people, especially after the age of forty years; it produces osteophytic outgrowths from the bodies of the vertebræ, but not ligamentous calcification and bony ankylosis. Various authors have adopted different names for this disease, the most frequent being spondylosis or arthrosis, because its ætiology has been considered to be degenerative and the result of age.

Spondylitis ankylopoietica has been described by many writers, particularly von Bechterew, Strümpell, Pierre Marie and Franckel. The name of von Bechterew was associated with that variety of the disease which commenced in the cervical region and extended downwards, whilst the other type, described by Marie and Strümpell and named *spondylose rhizomélique*, began in the lumbosacral area and extended upwards, producing ankylosis and the typical "poker back". It is now conceded that the disease is the same whether it attacks initially the upper or lower area of the vertebral column.

There has crept into American rheumatological literature the term "rheumatoid arthritis of the spine" (rheumatoid spondylitis), because the rheumatologists of the United States of America (as judged by their writings) regard *spondylitis ankylopoietica* as the spinal form of rheumatoid arthritis. Buckley[1] considers that ankylosing spondylitis is a disease *sui generis* and not a form of rheumatoid arthritis. Gilbert Scott[2] doubts whether it should be included in the rheumatic group at all.

The various names given to these diseases in different countries, denoting either some particular feature or some early investigation, undoubtedly add to the complexity of a subject already ætiologically abstruse and point to the need for a common international nomenclature. However diverse the various labels of *spondylitis ankylopoietica*, they nearly all reveal the outstanding features of the disease—namely, ankylosis and ligamentous calcification—and thus the significance of the disease, its course and final picture are widely recognized.

Gilbert Scott's reason for the term "adolescent spondylitis" is based on his conviction that "the pathological process which causes the condition known as ankylosing spondylitis always begins in early adult life, in spite of the fact that symptoms may be delayed until late middle age".

Freund recommends differentiation between primary and secondary *spondylitis ankylopoietica*, the former coming on insidiously and slowly without evidence of infection, and the latter following infectious diseases such as rheumatic fever, septicæmia and gonorrhœa.

History.

The earliest description was supposed to have been given by O'Connor in 1691 (quoted by Scott). The disease may have attacked ancient animals, as examination of fossils of the sabre-toothed tiger reveals characteristic signs of spondylitis, and Ruffer (quoted by Scott) found evidence in a dinosaur and crocodile of the lower Miocene period. Egyptian mummies give evidence of the disease as early as 5000 B.C., and Ruffer reports that one spine had been converted into one solid block.

This observation is interesting, as it proves the disease occurred in a hot, dry climate, and when foods were consumed before the advent of the modern manufacturing methods. It is therefore not a product of modern civilization. Freund,[3] however, states that it is only a few decades since "*spondylarthritis ankylopoietica*" has been recognized as a morbid entity and separated from the degenerative changes of the spine.

Pathology and Histology.

Freund states that the pathological literature is rather scanty, most of the reports being based on dry, macerated specimens. He gives a list of six histological examinations in this subject from Siven (1903) to Klinge (1934). He states that "Siven as well as Guntz (1933) found inflammatory changes in the synovial membrane, capsule and ligaments; therefore, they regard the condition as inflammatory". Freund examined a specimen of the lumbar vertebræ of a man, aged sixty-seven years, who had given a history of pain and stiffness of six years' duration. He states that the X-ray picture showed the "bamboo" appearance typical of ankylosing spondylitis. There was an absence of calcification of the anterior and posterior longitudinal ligaments of the spine. He mentioned that some authors stress the involvement of the intervertebral and costo-vertebral joints, while others stress the ossification of the ligaments. In the case he reported, the sacro-iliac joints had not been examined radiographically and were unavailable for pathological investigation. Availability of these joints for both examinations would have been invaluable, in consequence of Gilbert Scott's stressing of them as the joints primarily involved.

The specimens examined by Freund presented all gradations of fibrous and bony ankylosis of the intervertebral articulations. A pannus formed from the synovial membrane had caused the absorption of the articular cartilage, its replacement being by fibrous tissue. Investigation of the disks revealed the replacement of the cartilage and *nucleus pulposus* by invading blood vessels and fibrous tissue, the latter showing evidence of cartilage formation. Ossification of the ligaments was found in a commencing stage only. There was no involvement of the cord or intervertebral foramina, which is extraordinary when one considers how often coughing produces pain in the thoracic area and down the thighs in disease of the lumbar part of the spine. Discovery of the pathological changes in the apophyseal joints was valuable, especially as an opinion is current in medical literature that the disease begins in these joints, whereas Scott stresses the initial involvement of the sacro-iliac joints, at any rate from the radiographic point of view. Jacques Forestier[4] states that "it is generally accepted that it begins as an atrophic arthritis of the apophyseal joints, with later ossification of the ligaments, flava anterior and posterior longitudinal and thus giving rise to the bamboo effect". He, however, stresses the sacro-iliac involvement. A search of the literature fails to reveal any histological examinations of the sacro-iliac joint in the early stages of the disease, so that all the evidence for its primary involvement must come from a radiological source. Plenty of specimens are available exhibiting the final stages of the disease, when the picture may reveal bony ankylosis of the sacro-iliac and apophyseal joints, calcification of the anterior and posterior longitudinal ligaments and the *ligamentum flavum*, decalcification of the vertebræ in the involved area, the last-mentioned being so soft in some instances that they can be easily cut with a knife, and finally, an extension to the shoulder and hip joints and in a few cases to the remaining joints of the body.

Buckley[5] states that "an early, probably the earliest pathological evidence of the disease is decalcification generally in the bones of the pelvis and lumbar spine". Gunnar Edstrom[6] states that in ankylosing spondylitis the pathological process consists of chronic synovitis and tendinitis with round-cell infiltration, hyperæmia and proliferation of the connecting tissue.

Lloyd Tyson[7] describes the pathological process as a primary synovitis of the intervertebral joints with subsequent calcification of the ligamentous structures and eventual rigidity of the spine. He notes that if the small joints are involved they resemble those found in typical rheumatoid arthritis, a fact also stressed by Goldfain.

Buckley[27] quotes Stockman (1926) as advancing the view that ossifying spondylitis, more usually termed ankylosing spondylitis, originates as periarticular fibrositis. Some support is given to this theory by the fairly common observation that pain, disability and limitation of movement may exist for years before the appearance of any radiological evidence of the disease.

Ætiology.

Nothing is known with any degree of certainty of the ætiological cause, and even the ubiquitous streptococcus does not find many advocates. Some suspect a tuberculous basis, and Gilbert Scott states that "I am convinced that tubercle will eventually be found to be the cause of spondylitis in a certain percentage of cases".

The occurrence of iritis and spondylitis is fairly common, and as tuberculosis is a common cause of iritis (in Europe), it has been thought that the tubercle bacillus may be the causative factor, or one of them. Ponndorf's vaccine was extensively employed in France, and this fact revealed the trend of opinion with regard to the possible ætiological relationship of tuberculosis. This vaccine was made in the following three grades: (*a*) a mixture of bovine tuberculin with an autolysate of Koch's bacillus; (*b*) the same basis with autolysates of streptococci, staphylococci, pneumococci and the influenza bacillus; (*c*) the same, with the addition of gonococci.

The belief that this disease can follow gonorrhœa is well authenticated. We have encountered one case in which it followed immediately after the infection and within eighteen months had involved the whole spine and every joint in the body. This case occurred before the advent of the sulphonamides. This type of case gives support to an infectious basis, but still adds to the complexity of the subject of ætiology, as only a few cases, at any rate in this country, could possibly have a gonococcal origin.

Forestier states that in 60% of cases investigated by him and his confrères a genito-urinary ailment had occurred as a past history and was apparently occasioned by the gonococcus, by the *Bacillus coli communis* or by some other infectious germ. He states that "we believe the primary focus to be in the genito-urinary system or lower part of the bowel and that the toxic products are drained into the lymphatic vessels of the pelvis and along the spine". He points out that the lymphatic vessels from the prostate and seminal vesicles pass on both sides of the medial line in front of each sacro-iliac joint and along the posterior surface of the abdomen on both sides of the spinal column just in front of the apophyseal joints. They are directly applied on the vertebral column behind the aorta and *vena cava*, and they have few anastomotic connexions with the rest of the retro-aortic system. These facts may explain the slow progression from the sacro-iliac joints and the ascent of the lesion along different segments of the spine.

Septic foci in teeth, tonsils *et cetera* may be responsible for some cases; but the removal of septic foci has been followed by no dramatic recoveries, such as may occur in a number of infective conditions and in rheumatoid arthritis.

Gilbert Scott[8] records the negative result of bacteriological examinations of adjacent bone surrounding the

sacro-iliac joints removed by open operation and trephine under local anæsthesia. Robinson[9] tested for tuberculous sensitivity 45 patients suffering from ankylosing spondylitis and concluded that, in comparison with 90 patients with ordinary rheumatic conditions used as control, the spondylitic group showed a 30% higher sensitivity.

Steinbrocker[10] states that "specific infections such as gonorrhœa at times precede the appearance of a rheumatoid spondylitis, but any direct relationship still remains to be proved".

We have under therapy at present four patients suffering from typical ankylosing spondylitis, whose disease began almost immediately after a gonorrhœal infection. Clinically and radiographically their condition does not differ from that of patients with no antecedent Neisserian history. The blood serum of all four fails to react to gonococcal fixation tests, and one had chronic prostatitis with large numbers of pus cells in the smear of prostatic fluid examined, but no gonococci. Sulphanilamide therapy and vasotomy with the injection of 10 mils of a 2% solution of mercurochrome in two cases and of penicillin in another as well as 500,000 units given intramuscularly, have not altered the disease in any way and not prevented the involvement of some large joints. One man, who contracted a Neisserian infection thirty years ago and has had typical ankylosing spondylitis, the whole spine being involved, has during the last twenty-eight years had four healthy children. The presence of a slowly advancing disease with no impairment of fertility would almost rule out the continued activity of a gonococcus and any possible ætiological relationship.

Goldfain[11] reported eighteen cases of chronic brucellosal type of ankylosing spondylitis. It was observed that males were affected more commonly than females at a ratio of eight to one, and that the lumbar area of the spine was the most common region for localization. Goldfain makes the following statement:

> The symptoms of ankylosing spondylitis as a complication to chronic brucellosis are those of a case of infectious spondylitis in which pronounced rigidity of the spine, either locally or throughout its entire extent, plus either local or diffuse muscular spasm, will be present. Rigidity of the spine is especially present if the intervertebral articulations are involved.

Goldfain gives three distinct phases of involvement: (i) prespondylitic—inexplicable symptoms; (ii) sacro-iliac involvement; (iii) pronounced spinal rigidity. He remarks that the X-ray findings are positive in the second and third phases, but not in the first. Our knowledge about the ætiology would, therefore, seem to be in a confused state, and it is possible that the disease may be caused by a number of different agents.

Buckley states that the cause is unknown, but is undoubtedly infective. He thinks that the gonococcus is certainly responsible for some cases of arthritis of the spine, but in the light of modern knowledge it is doubtful whether it causes ankylosing spondylitis. He states that when gonorrhœa has been present, involvement of the smaller joints precedes that of the spine; but in true spondylitis the progress is always from the spine to the large joints in a regular centrifugal course, and although the small joints may be involved early, there is a remission in the disease when the spine is affected. He notes that spinal injury appears to be a factor, and we have encountered a number of cases in which the symptoms followed a recent injury to the spine.

In a number of cases in our series the history of arthritis following a gonococcal infection, in various joints, with the subsequent concentration in the spine to the exclusion of other joints, has been obtained.

Although some authorities believe that tuberculosis may play a part in the ætiology of ankylosing spondylitis, it is wiser to disregard it as an important factor, especially from the point of view of projected therapy, because immobilization in a plaster cast, which is beneficial and necessary in spinal tuberculosis, is fraught with many tragedies of marked acceleration and spread of the disease. Whatever the ætiology that may finally be determined in this disease, prolonged plaster immobilization is detrimental and should never be undertaken.

Incidence of the Disease and Age of Onset.

The disease is commoner in males than in females. It is generally agreed that it occurs from six to ten times more frequently in men than in women. Tyson notes that it often affects two or three members of the same family. We have at present a family in which three members are affected. Scott, who has collected the greatest number of cases, 555 having been investigated during nine years, reveals that 396 of the patients were males and 159 females. Swain treated 106 patients over a period of twenty years, the ratio being four to one in favour of the males. Hare[12] states that the disease is ten times more frequent in men than in women; he remarks that the disease may begin between the ages of twelve and twenty-five years. Blair noticed that pain may begin at the age of twelve years. Scott lays stress on the prespondylitic stage and the symptoms occurring in early life, and his title of adolescent spondylitis is descriptive of his view.

All writers stress the fact that the disease attacks the vigorous, healthy people, and, according to Scott, amongst athletes, swimmers are particularly prone to it. We, however, have not noticed this latter preferment in New South Wales. The observation that the disease attacks the strong and vigorous seems to be an important point against incriminating the tubercle bacillus as a frequent causative factor. The average age in Buckley's series was thirty years.

Tyson[7] states that at the Presbyterian Hospital one case is encountered in every thirteen cases of rheumatoid arthritis.

The incidence of the disease in young, healthy males is of paramount importance, not only from the point of view of suffering, but also in relation to decreased capacity for work, to chronic invalidism and to the resultant economic loss and cost to the community. Because of the incidence of ankylosing spondylitis in young, healthy, adult males and the frequency of rheumatoid arthritis in young females, these diseases assume a position of national importance, as they rob the State of some of the potential parents in an age group at the very beginning of family life. Any measures designed to control and thwart the disease in its relentless onward march should be invaluable to the State and to the people unfortunately afflicted.

In our series, although some subjects were athletic, others hard-working farmers *et cetera*, there were a number who gave no history of sports activity, and one was a musician with only moderately strong physique. Apparently, at any rate in Australia, the athletic and non-athletic are affected, no pronounced stress being laid on the former. In 41 cases encountered before 1938, a preponderance of three to one in favour of males was observed.

Climate and Moisture.

With regard to climate, the disease occurs in the higher mountain altitudes, in the drier interior and in the coastal high rainfall belt. It is seen in the hot, dry area inland and in the hot, moist conditions in northern New South Wales, and one patient at present under our care developed the disease in New Guinea during the wet monsoonal weather. The disease in this man was active and rapidly progressive.

Mode of Onset; Symptoms and Course of the Disease.

Most authorities are agreed that prespondylitic symptoms occur in many cases. In the typical case the patient may have had aches and pains in various areas of the body without definite localizing signs. In one case of our series the metatarsal joints had been affected for years, the result being collapse of the metatarsal arch and clawing of the toes. These joints had been treated for a few years before any complaint was made concerning the

back, and a radiographic examination of the sacro-iliac joints then revealed early arthritis of the joints of the ankylosing variety.

In many cases the condition is so insidious in its onset that although the patient complains of a sudden attack of back pain, an examination reveals a typical "poker back" and ankylosis. In other cases the initial symptoms and signs resemble those of acute sciatica, and in many cases sacro-iliac involvement is masked by the pain along the distribution of the sciatic nerve. One of our early patients has been watched over a period of fifteen years, his initial symptoms being pain along the distribution of the left sciatic nerve. The diagnosis of sciatica was retained for years, because radiographic examination of the spine revealed no lesion; but later a "poker back" developed, and the disease finally invaded all the spine. A number of patients seem to develop symptoms and signs in the thoracic area, pain and restricted breathing heralding the onset. This type of onset is often associated with cervical signs. The variegated pattern of symptoms and signs must be obvious when one considers the widespread nature of the spinal involvement, and the initial symptoms and signs must be dependent on the areas first affected.

Often the pain in the lumbar region radiates down the back of both thighs as far as the knees, and such a distribution in a young man or woman should immediately raise in our mind the possibility of ankylosing spondylitis and call for careful examination of the sacro-iliac joints. In one of our cases the disease first attacked the lumbar area and after a few months left that situation and attacked the cervical and dorsal area, no radiological evidence of sacro-iliac involvement being obtained.

Although the insidious and slow onset of the disease is frequently stressed, and although this factor accounts for the failure to make the correct diagnosis in many cases till the tragedy of ankylosis is discovered, the variety characterized by rapid onset and progress of ankylosis of the whole skeleton is often seen (10% incidence according to Tyson), and we can recall one case in which every joint was rigid in two years, with the exception of the temporo-mandibular joints. The fingers assumed a bizarre shape, resembling various forms of stalactites. Happily these severe, fulminating lesions are rare, but even when they are treated early they relentlessly pursue their course undiminished by therapy. Only if these migratory and minimal symptoms and signs in the prespondylitic phase are given their rightful place and a correct perspective is maintained will many patients be saved from entering the dark valley of extensive spinal ankylosis.

In the prespondylitic stage of the disease it is the distribution of the vague pains in the extremities without spinal involvement which is commonly seen, and in consequence of this the serious nature of the malady is not realized. The abdominal symptoms and signs are sometimes severe enough to be diagnosed as gall-bladder disease, gastric ulcer or even appendicitis, and appendicectomy has been performed in consequence of an erroneous diagnosis. In this stage the symptoms may disappear for years or months, and this quiescence gives a feeling of false security and seemingly verifies the glib and soothing diagnosis of muscular rheumatism.

It is in this prelude period that the rule of the British Red Cross and Charterhouse Clinics is invaluable—namely, "the sacro-iliac joints are to be examined radiographically in all patients between the ages of 14 and 25 years who have complained over a period of years of recurrent rheumatic pains". Some writers have testified to the greater number of cases diagnosed after radiographic examination of the sacro-iliac joints, when symptoms and signs are indefinite and indeterminate.

Tyson quotes Krebs as stating that 40% of his patients suffered from pains resembling sciatica.

Morning stiffness, which disappears on arising and moving around, but which may recur if rest is taken during the day, is a characteristic symptom, and may be localized to the lumbo-sacral area of the back. In such a case radiographic evidence in the sacro-iliac joints may even be wanting, but there is some suggestion that decalcification may occur at this stage and indicate the likelihood of the malady, the confirmation of the diagnosis being dependent upon time and upon serial X-ray examinations.

When pain and stiffness are becoming localized in a particular area of the spine, the possibility of this disease must be borne in mind, even if a normal blood sedimentation rate is present. Fever may occur in the acute stage, but rarely do any patients have a temperature above 99° F. at night. In the second stage there are generally four cardinal symptoms and signs: rigidity, deformity, affection of the root articulations, and sensory and motor changes due to pressure on the nerve roots.

The attack of wandering pains may now disappear and be replaced by pain and stiffness localized to a portion of the spine, the pain being variable in severity even at this stage. One of the patients with the most severe pain we have encountered was aged between fifty-five and sixty years, and one complaining of the least pain, even in the presence of active progressive disease, was aged twenty-two years. Thus age does not determine the degree of disease activity or the severity of pain.

Rigidity in the lumbar area is easily diagnosed if the patient is asked to touch the toes with his hands whilst his knees are fully extended, but thoracic rigidity is easily missed unless specifically sought. As the disease advances, spinal rigidity may increase fairly rapidly and be evidenced by considerable diminution of back flexion; but it is often remarkable to see the degree of spinal rigidity that can be present while the patient's hands can still touch the toes when his knees are extended. In some cases radiographic films may reveal very little abnormality, whilst reduction of movements is pronounced, the severity of the clinical condition being disproportionate to the radiological picture. The rigidity may, therefore, be due to fibrous ankylosis, and this is well exemplified in Figures I and II. The X-ray picture revealed little bony change, no ligamentous calcification having occurred, and this was not evident until three years later.

Restriction of movement is generally more pronounced if the disease has primarily attacked the lumbar and sacro-iliac areas than if the main weight of the disease has fallen on the dorsal part of the spine. Not only are flexion and extension reduced, but rotation and lateral flexion are much diminished.

In the cervical area considerable rigidity may be present, only a slight nodding movement remaining. Sometimes the chin will be drawn down on the chest, and a photograph from the posterior aspect now reveals a "decapitated" patient, owing to the severe deformity of pronounced dorsal kyphosis and extreme cervical flexion.

Deformity.

The deformity is so characteristic that in many cases the diagnosis can be made at a distance. The neck stiff and craned forward, the dorsal kyphosis and the slight flexion deformity of the hip joints give a typical picture. On examination of the patient, the lumbar area may be completely flattened, a "poker back" or lordosis remains, and the sacro-spinalis stands out taut on either side of the spine, the sulcus between the muscles being deeper than normal. In some instances slight scoliosis is present, and rarely, the normal lordosis is replaced by a kyphosis. The condition of the cervical area may not only be affected by a craning forward, but in some instances some degree of rotation often combined with lateral flexion may be present. The extreme flexion deformity of the cervical area as revealed in Figure I not only indicates the severity of the disease, but also bears witness to the complete lack of preventive therapy which allowed its development. On frontal examination the flattened chest, the forward carriage of the shoulders and an abdominal groove at or just above the umbilicus may be present. Scott has described a typical spondylitis facies, but this is seen only when the neck is craned forward and almost rigid.

Affection of the Root Articulations.

Pain on coughing and sneezing or on stepping down even ordinary steps is sometimes severe, and many of these

patients are unable to sit squarely on a hard seat, complaining that the pressure on the buttocks produces pain down the back of the thighs and even to the calves of the legs. The hips are much more frequently involved than the shoulders, and flexion deformity is easily discerned as the patient attempts to assume the upright position. Abduction and rotation become limited as the disease becomes more active in the joints. If the disease advances, complete bony ankylosis of the hip or hip joints may result, and this may occur even if the patient is ambulatory and refuses to be confined to bed even in the acute stage. The shoulders are often affected by limitation of movement, internal rotation and extension of the arms above the head being commonly lost; but ankylosis is extremely rare. The spread of the disease is generally centrifugal, the root articulations being involved after the spine; but this rule does not always follow, as we have seen a knee and elbow involved without involvement of the hip or shoulder. Although the knee joint may become ankylosed when the hip joint has suffered a similar fate, in some cases the former joint may be affected by severe synovitis with a distended capsule owing to the presence of a large quantity of synovial fluid with or without a number of polypoid synovial fringes palpable or discernible by movement. If the fingers and wrist joints are involved, the appearances may simulate those of rheumatoid arthritis of the same areas; but we have never observed the pink finger tips, skin pallor and distended volar venules so commonly observed in this disease amongst females of the child-bearing age.

Sensory and Motor Changes.

Pressure on the nerves at their exit from the spinal canal may produce radiating pains, even neuritis, the nerve being tender and painful and some areas of anæsthesia being present along the limbs.

It is well known that in some cases sciatica diagnosed as due to *nucleus pulposus* is caused by pressure of an inflamed and adherent *ligamentum flavum*. As this ligament is involved in the calcification so commonly seen in this disease, it is possible that the frequently concomitant sciatica may be due in many cases of ankylosing spondylitis to its inflammation. Muscle spasm is generally pronounced, and the slightest amount of movement may initiate a cramp-like contraction, causing the patient to cry out with pain. The knee jerks are generally exaggerated, and the slightest attempt to initiate them may be accompanied by severe pain and muscular spasm, the patient becoming apprehensive at the slightest attempt at movement. Muscular wasting is also generally pronounced if the disease is widespread.

In its final stage the disease may involve only a local spinal area, or all the spine with or without root articulations, or the entire body, even movement of the jaws being greatly restricted. The variation in the intensity and the different degrees of involvement result in a varied pattern of the disease. In some cases it pursues its course relentlessly, but in others Nature calls an effective halt, which may last for many years or for life. Exacerbations with further extension are common, especially after an attack of influenza or the common cold.

Death may occur from intercurrent infection, and pneumonia is a common terminal event, as one would expect owing to the reduction in chest expansion.

Iritis.

Iritis may occur at any stage. It was thought by some to point to the possibility of a gonococcal infection as the *fons et origo* of the disease; but it often occurs without any antecedent history of Neisserian invasion and in the presence of completely negative results to tests for the gonococcus. The disease may be acute and severe, rapidly producing adhesions and calling for prompt treatment by an ophthalmologist if irreparable damage is to be avoided.

French authorities on ankylosing spondylitis have stressed the association of tuberculosis as a possible ætiological factor in iritis, but support for the tubercle bacillus or the gonococcus as causative factors has been wanting in most other centres of research. The association of iritis and spondylitis would seem to be more frequent than the association of iritis and rheumatoid arthritis.

Thyreoid Involvement.

Thyreoid enlargement has been observed in a small number of cases of this disease; but as the neck is rigid in the advanced cases and craned forward, the prominence may be due mainly to this abnormality. In one case the basal metabolic rate was +22%, and examination after complete rest in bed revealed a constantly rapid pulse and night sweats. Night sweats, a rapid pulse and a slight nocturnal rise in temperature of about 1° F. are often present in these cases in the absence of any evidence of enlargement of the gland.

The Parathyreoids and Calcium.

Investigation of the serum calcium content shows very little deviation from normal, even in the presence of pronounced decalcification. Gilbert Scott has shown that in contradistinction to rheumatoid arthritis, which produces a general skeletal decalcification plainly visible in a standard X-ray film of the hand, there is no evidence of a similar process in the hand of the patient suffering from ankylosing spondylitis, unless it is also involved in the disease. He inclines to the belief that there is a local but not a general systemic decalcification in the latter disease, and this constitutes an important point of difference between the two diseases.

A few years ago parathyreoidectomy was practised by some Russian and later some American surgeons as a curative measure in ankylosing spondylitis. The parathyreoids are enlarged in this disease, as they are in most infections, and it was thought that this operative procedure might prevent decalcification and cut short the disease. The Russian authors were enthusiastic and published results of over 70 cases; but after the first waves of American enthusiasm had reached the shores of critical analysis and later careful biochemical investigation had proved the absence of a negative calcium balance, the advocacy of the procedure died a natural death.

Leriche and Jung had published results obtained in a number of cases in which the former had operated, and in some of which the parathyreoids were absent on microscopic examination, portion of the thyreoid alone being removed; but time did not substantiate the early optimism of these outstanding investigators.

We checked the serum calcium content of a large number of patients suffering from ankylosing spondylitis and rheumatoid arthritis without finding any great variation from normal.

Ankylosis.

Ankylosis may be fibrous or bony, and the former may remain for a variable period of years before bony union occurs. Some ankylosis apparently remains in this fibrous stage without proceeding to that of bony ankylosis. This was well demonstrated in the case characterized by severe dorsal kyphosis and cervical flexion, in which, owing to the fibrous stage of the union, correction of much of the deformity by manipulation was possible. Three years later bony ankylosis and ligamentous calcification were much in evidence. It has been pointed out that the ankylosis of the apophyseal joints in the lumbar area accounts for the great diminution in movements about that area, and it is remarkable how little diminution of movement results from bony ankylosis of the sacro-iliac joints if these other joints are not involved.

Pathological Tests.

The pathological tests of value are a red cell and leucocyte count, a test meal examination, estimation of the blood sedimentation rate, the hippuric acid excretion test, and the estimation of the plasma protein content. The most valuable test to gauge the activity of the disease is the estimation of the blood sedimentation rate, and the result

generally parallels the severity and stage of the disease. It is not an infallible guide to the degree of activity, and in some of our cases there has been slow yet progressive increase in the disease in the presence of a normal blood sedimentation rate.

A slight degree of anæmia and an increased leucocyte count are often present, especially in the acute stages. The facial appearance is no guide to the presence or absence of anæmia, and it is remarkable how often healthy outward appearances conceal a degree of anæmia. Although an increase in the white cell count and a "shift to the left" are often present in the acute stages, a much raised blood sedimentation rate may accompany a normal leucocyte count. Test meal examinations should be a routine measure, as the addition of hydrochloric acid may help alimentation by increasing appetite and aiding digestion.

The hippuric acid test may be of value in revealing a reduction of liver detoxicating capacity by glycine conjugation, but the result is often normal in the presence of a much increased blood sedimentation rate.

The estimation of the plasma protein content demands much time and is costly; but both in ankylosing spondylitis and in rheumatoid arthritis the test may reveal a great reduction in plasma albumin and an increase of globulin (especially euglobulin) and in fibrinogen. The total protein content may be altered only slightly or not at all; but the reduction of plasma albumin content may lead to a lowered albumin-globulin ratio (1:1 or even lower). This will account for the non-cardiac and non-renal œdema which characterizes so many of the severe cases of both the above-mentioned diseases. The increase of fibrinogen is generally greater in ankylosing spondylosis than in rheumatoid arthritis, as has been noted by Aldred-Brown.[12]

The blood phosphatase level is not raised to any great extent, and this is in contrast to malignant disease of the spine and Paget's disease. In difficulties of diagnosis increased values may point to the presence of concurrent malignant disease.

In a group of 41 patients examined and treated before X-ray therapy was begun in 1938, there were some interesting deductions: achlorhydria was present in 28% of cases, hypoacidity in 25%, normal acidity in 38%, and hyperacidity in the remainder. A disturbance of gastric secretion towards hypoacidity and achlorhydria occurred in a little more than half the number of cases.

In approximately 50% of the group "a shift to the left" of the neutrophile cells was present, but only six patients had a white cell count in excess of 6,000, and two had a count of 4,000 or less.

Approximately 50% had a raised blood sedimentation rate, over 12 millimetres at 60 minutes (Westgren), the highest figure being 58 millimetres.

Diagnosis.

Gilbert Scott lays stress on the prespondylitic symptoms in the young, active male and female, emphasizing the importance of early radiological examination of the sacro-iliac joints. The different modes of onset are important and should be kept in mind, and the evanescent and wandering pains, which may be everywhere except in the lumbar and sacral area, often obscure the true picture. Sciatica is a common antecedent, but many cases of this disease do not terminate in ankylosing spondylitis. One of the strange features of the disease is to find that a large joint like a knee joint has been involved for a long period previous to the localization of the lesion in the back, but after localization has occurred the joint disability disappears entirely. This localization of the disease in the spinal column followed by complete subsidence in a large joint has been pointed out by many authorities.

In some cases rheumatoid arthritis of the upper limbs is accompanied by cervical spondylitis. In these cases the sacro-iliac joints are frequently normal.

It is the type of patient, all too frequently encountered, who complains of acute backache resembling lumbago and in whom there is no radiological evidence of joint involvement, who may cause a diagnostic error to be made by the physician who does not keep the disease in mind or has not followed some such patients through the years. In one such case in our series there was in 1938 no radiological evidence of the disease in the sacro-iliac joints, but pain in the chest necessitated in 1942 an X-ray examination for tuberculosis with negative results; it was not till early in 1944 at a general examination that we found a "poker back" and diminished chest expansion, whilst radiologically the sacro-iliac joints were affected by severe inflammation, and there was some early calcification of ligaments in the dorsal and lumbar areas of the spinal column.

Scott states that in these cases an erroneous diagnosis has led to surgical intervention in the abdominal cavity, without relief of symptoms, which are due to spinal involvement. The absence of signs of peritoneal irritation should facilitate diagnosis. In cases in which chest symptoms resemble those of pleurisy, the fixture of the thoracic cage on both sides and the history of low back pain should provoke thoughts of the possibility of ankylosing spondylitis. We are of the opinion that in many cases the disease is one of fibrositis of the spinal ligaments leading to deformity and reduced movement, without radiological evidence for a long period of any bony abnormality such as ankylosis or ligamentous calcification. This aspect was well presented in the case from which Figures I and II are taken; the disease began when the patient was aged

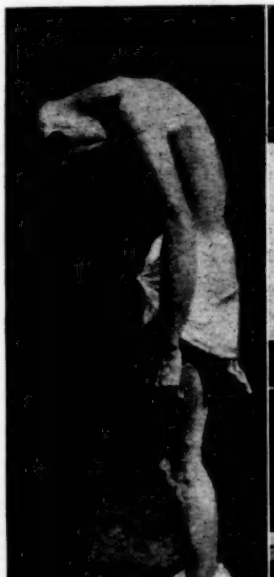

FIGURE I.
Fibrous spondylitis, before manipulation.

FIGURE II.
Fibrous spondylitis after manipulation performed by the late E. B. M. Vance.

twenty-seven years, and in three years it had produced the appalling degree of cervical curvature depicted in the photograph. It was only the fibrous nature of the disease that enabled the manipulator (the late Dr. E. B. M. Vance) to produce the dramatic reduction in the deformity.

Scott has laid great emphasis on the fact that in the early stages, when pains are distributed throughout the body, an early non-symptomatic sacro-iliitis may be in

progress, whose silence often leads to many diagnostic errors and whose presence may be suspected only when a typical "poker back" is discovered on routine examination. He has also asserted that "the amount of calcification in relation to the severity of the disease is variable", and further, that "complete rigidity of the spine may be present without any calcification of the ligaments, and unaided by an X-ray examination it is impossible to determine whether this rigidity is mechanical or spasmodic; even though calcification may be absent even in an advanced case of spondylitis, the sacro-iliac joints are always abnormal".[25]

FIGURE III.

Case XXII. The whole spine is involved. Note the "poker back", dorsal kyphosis, the craning forward of the neck, the abdominal groove at the level of the umbilicus.

FIGURE IV.

Case XXX. The whole spine is involved except the cervical area. Note the flat back and severe reduction of back flexion. The patient had been a fine, athletic type. In spite of the photographic evidence of great reduction of movement, the blood sedimentation rate was normal (3·0 millimetres at 60 minutes).

Hare[12] has stated that "at the Lahey Clinic they have been more conscious of the disease both clinically and roentgenographically and have diagnosed more than 35 cases in three years, and it is a larger group than had ever been diagnosed in the clinic prior to this time". Forestier states that the clinical picture is often confusing and therefore regrettable, as the condition is controllable if diagnosed early. He states that purely fibrous adhesions may be present for some time before bony ankylosis takes place.

The diagnosis is easily made in the advanced and severe cases. A complete examination of the whole spine as a routine measure is absolutely necessary in all suspected or doubtful cases, and it is the failure to make this examination that leads to errors. Investigation of the spine may reveal a "poker back", a flattened lumbar area, kyphosis of the dorsal vertebræ, and stiffness and forward craning of the neck. All movements of the lumbar part of the spine may be much diminished, back flexion being so limited that when the patient's knees are extended the outstretched hands will not reach to the level of the knees. This great reduction of movement may occur even when little radiographic abnormality is apparent, and conversely, radiographically advanced disease may be associated with a surprisingly good range of back flexion. It has been stated that the greatest reduction of range in movement occurs if the apophyseal joints are involved, and undoubtedly when calcification occurs reduction is considerable.

The sacro-spinalis muscle may be flattened on either side of the mid-line, but at times the muscles stand out on either side with a deep sulcus between them. In some cases a slight lumbar scoliosis is present and in a few lumbar kyphosis is found. The thoracic area may be affected by slight or pronounced kyphosis.

The neck is generally craned forward if it is involved, and may in addition be affected by scoliosis and rotation. The movement of the head may be reduced to a mere nodding. In the cervical part of the spine severe flexion deformity may also be found, the chin almost touching the chest.

If the costo-vertebral joints are involved, reduction of chest expansion may be pronounced and pain may occur on coughing, sneezing or exertion.

In the abdomen there is frequently a transverse groove at the level of the umbilicus caused by the dorsal kyphosis in association with the "poker back".

Muscular wasting of spinal, shoulder and gluteal muscles is common. The muscles are often irritable, the exaggerated reflexes requiring only the slightest touch for their elicitation.

Shoulder and hip involvement may lead to restricted movement and at times to ankylosis of the hips.

In some rare cases almost every joint of the body, including the jaw, becomes completely or partially ankylosed, and in two cases we have seen complete ankylosis of every joint except the jaws.

The hands may be cold and clammy, but they never have the pink finger tips and distended phalangeal veins with atrophic skin which are common in rheumatoid arthritis.

X-Ray Diagnosis.

There is still some difference of opinion as to whether the initial radiological evidence of the disease is to be observed in the apophyseal or sacro-iliac joints. Scott lays great emphasis on the sacro-iliac joints as the initial joints to be involved, whilst Tyson states that "if commencing in the lumbar area synovitis of the sacro-iliac joint is the first sign, but if in the thoracic a costo-vertebral synovitis is the earliest change". Hare states that sacro-iliitis is the first evidence of the disease, and suggests that an X-ray picture should be taken at a 15° angle view, the patient being supine, with knees flexed, and the X-ray tube being tilted 15° cephalad.

Blair[14] states that the first abnormal change takes place in the sacro-iliac joints.

Forestier makes the statement that "it is generally accepted that the disease begins as atrophic arthritis of the apophyseal joints". He observes that early diagnosis by X rays is often not helpful, because the appearances in the apophyseal joints are difficult to interpret radiologically. In further discussion he intimates that "they have found the sacro-iliac joints show radiological changes early and are of enormous value in the diagnosis of ankylosing spondylitis". He points out that Pierre Marie and Levé had reported the ankylosis in the sacro-iliac joint in their first description of the disease, but only in the last decade have radiologists noticed that this fusion was associated with the "bamboo" spine. In 153 cases Forestier found only two in which the sacro-iliac joints were not involved in ankylosing spondylitis.

Oppenheimer[15] points out that oblique views of the spine show the *ligamentum flavum* and lateral fibres of the longitudinal ligaments, and states that the first lesion in *spondylitis ankylopoietica* is an apophyseal joint lesion, and that the *ligamentum flavum* may be associated with the inflammation by adhering to the capsule of the joint. The X-ray changes in the sacro-iliac joints have been well summarized by Gilbert Scott, who stresses the difficulties encountered in the early stages when radiological evidence

is slender and its interpretation difficult. He describes three stages:

First Stage: The first stage is characterized by loss of definition of the fissures, by widening or blurring of the joint or by mottling of the cartilaginous section.

Second Stage: The usual well-defined, narrow, pear-shaped linear shadow becomes broad and indefinite and the fissures are obliterated. There may be small, rounded cavities giving a rosary effect, and these may coalesce.

Third Stage: The third stage is characterized by obliterated fissures—irregular areas of rarefaction, osteosclerosis in the surrounding bone if resistance is high or osteoporosis and decalcification if resistance is low, and later complete ankylosis.

Scott regards sclerosis of the bone as evidence of probable resistance of the patient, and its absence as evidence of low resistance and an unfavourable prognosis. He believes that decalcification in these areas is evidence of a high degree of virulence and an unfavourable sign. He lays stress on the observation that decalcification (osteoporosis) is confined to the areas of the body affected and is not generalized as in rheumatoid arthritis. The apophyseal joints can be examined in the usual antero-posterior or oblique view.

Some authorities state that the oblique view is necessary for a complete picture of the joints, and also that this view may reveal calcification of the lateral fibres of the anterior longitudinal ligament, which will not be detected by the orthodox antero-posterior and lateral views. Oppenheimer, who has carried out extensive investigation of the oblique views of the spine, states that involvement of the lower thoracic, apophyseal and costo-transversal joints may occur years before the sacro-iliac involvement. The usual changes are irregularity of the surfaces, sclerosis of surrounding bone and finally ankylosis with obliteration of the joint.

Forestier[4] describes the sacro-iliac changes in three stages: (i) pseudo-widening of the joint space, which may take several months or years, marginal decalcification in one or both joints generally, initially in a portion and usually in the lower segment of the joint; the margins, especially the iliac margin, are usually woolly and hazy; (ii) pycnotic formation in the joint area; a mottled appearance of the cancellous bone in both the sacrum and the ilium; an area of decalcification and hypercalcification (rarefaction and sclerosis); (iii) total synostosis with fibrillary ossification and areas of sclerosis; at this stage calcified ligaments of the spine are generally seen. He found that the best picture of these joints was obtained in the antero-posterior view, the knees being flexed and the thighs in abduction.

With regard to the other areas of the spine, flattening of the lumbar curve, scoliosis, thoracic kyphosis and increased flexion, hyperextension and rotation of the cervical area may be also observed radiologically.

The involvement of the longitudinal ligaments can be easily seen in the lateral or oblique views, and calcification is generally easily differentiated from osteophytic outgrowths. Forestier notes the difficulties in deciding whether the involvement is osteophytic or whether it is calcification (syndesmophytes) of ligaments or ossification resulting in bridging. However, in most cases it is easy to follow the line of the ligament, and the calcification is easily recognized. In severe cases the spine assumes the "bamboo" appearance and it is stressed that the interval between the vertebræ is not narrowed, as in *spondylitis osteoarthritica*.

The involvement of the *ligamentum flavum* may give the appearance of railway lines on either side of the middle line.

Oppenheimer[15] states that in 50 cases of Marie-Strümpell disease the *ligamentum flavum* was ossified in 22, the interspinous ligament in 12, and the longitudinal ligament in 25, and that fourteen months was the earliest time after the symptoms commenced at which ossification of the ligaments was noted. He states that ossification of the vertebral ligaments is a late and inconstant sign in the presence of Marie-Strümpell disease and other types of spondylarthritis (apophyseal arthritis).

The apophyseal joints are often obliterated early and the costo-vertebral joint may also be affected by ankylosis. The *symphysis pubis* is occasionally involved.

Although the X-ray picture makes possible verification of the clinical diagnosis and may partially reveal the degree of involvement, we would stress again that the absence of positive radiographic evidence in the early stages should not be regarded as disproving the presence of the disease.

There are two cases in this series in which the absence of positive X-ray evidence in the initial stages was followed within four years by pronounced and active disease obvious radiographically.

Differential Diagnosis.

Spondylitis Osteoarthritica.—The presence of osteophytes is diagnostic. The blood sedimentation rate and the plasma protein content are normal in practically all cases or are only slightly disturbed. The disease may follow an injury and generally occurs in middle and later life.

Spondylitis Associated with Inflammatory and Infective Diseases.—Acute spondylitis may occur during the course of typhoid fever and influenza. The vertebral body may become softened, later deformed and wedge-like. Staphylococcal infections may also attack the vertebral column and produce purulent osteomyelitis. Pott's disease may be mistaken in the early stages, but the X-ray picture will soon clarify the diagnosis. The history, X-ray appearances and temperature will also enable a diagnosis of the former diseases to be made.

Spondylolisthesis and Kümmell's Disease.—Spondylolisthesis and Kümmell's disease are easily differentiated by history and radiological findings.

Postural Deformity.—Kyphosis in the old and young is generally easily discernible and should present no difficulty. The intervertebral disks are involved and narrowing of the space takes place.

Secondary or Primary Malignant Disease of the Spinal Column.—In the absence of any positive radiographic evidence, cases of malignant disease of the spinal column present great difficulties in diagnosis. The pain of malignant disease and ankylosing spondylitis can be severe, necessitating morphine even in the early stages of both. The history of the removal of any antecedent growth may enable a tentative diagnosis to be made, and the age of the patient may incline towards the cancer period of life; but in the absence of positive X-ray evidence one cannot be certain. In one case the advanced years and severe pain favoured malignant disease, but time has proved that the patient was suffering from acute ankylosing spondylitis. The blood sedimentation rate is raised in both conditions, the plasma protein content is altered similarly; but in malignant disease there tends to be a much higher percentage of globulin than is generally present in *Spondylitis ankylopoietica*. There are two helpful points in the differential diagnosis when all other evidence is lacking: (i) The pain in malignant disease is generally more severe, reaches a maximum intensity rapidly and is apt to come on suddenly. It generally requires morphine, whilst the pain in ankylosing spondylitis rarely does. (ii) Heat, such as short-wave diathermy, will often precipitate an attack and severely increase the pain of malignant disease, but except when the spondylitis is accompanied by sciatica, diathermy will not increase its pain. One of us saw four patients within two years who had been diagnosed as suffering from spondylitis, and it was not till these two signs were present, and only after many months of careful watching, that the tentative diagnosis of primary or secondary malignant disease could be made.

Parkinson's Disease.—Parkinson's disease should present no difficulty, as the tremor and facies are diagnostic.

Osteitis Deformans.—If it is confined to the lower area of the spine, *osteitis deformans* may be confused clinically with *spondylitis ankylopoietica*, but the radiographic appearance of absorption and ossification—the cotton-wool appearance—is diagnostic.

Tuberculous Sacro-iliitis.—It is of the utmost importance not to confuse tuberculous sacro-iliitis with ankylosing

spondylitis, because the treatment is entirely different, even diametrically opposed, the former requiring and being relieved by complete long-continued immobilization, the latter being aggravated by similar therapy. This most important clinical fact will be discussed further under the heading of treatment. Tuberculosis of the sacro-iliac joints is comparatively rare, whilst ankylosing spondylitis is fairly common. Tuberculosis generally attacks one joint, and the pain is generally severe. A cold abscess over the joint or tracking down into the perineum may give a clue to the lesion's true identity. In the early stages tuberculosis is generally limited to the cartilaginous section of the joint, and the limits of the erosion of bone are well defined and suggest a cavity. Generally in spondylitis both joints are attacked simultaneously, one often more than the other. Tuberculous disease usually, but not always, attacks weaklings, whilst ankylosing spondylitis most frequently attacks the apparently healthy and often athletic type of subject.

Tuberculous Disease of the Hip.—Scott[16] makes the following statement:

> The most serious error in diagnosis that can possibly be made at this time is to confuse these symptoms of incipient spondylitis with those of tubercular hip-joint disease. Such a mistake has been the cause of much permanent crippling, which, as I shall show, can be avoided. Among the 300 cases of spondylitis investigated about ten had developed ankylosis of one or both hip joints in addition to fixation of the spine. Now in all these cases ankylosis occurred only after the joints had been immobilized in plaster for several months.

One of us has seen exceptions to this latter statement. In tuberculosis of the hip joints the sacro-iliac joints are normal; but whereas in spondylitis pain occurs around the hip joints, these joints are normal, but the sacro-iliacs show inflammatory changes. One of us has seen two cases in which plaster immobilization and long-continued dorsal decubitus resulted in generalized spread of the disease, the patient finally presenting the appearance of almost complete rigidity of the spine and upper and lower limbs. Although some cases will progress to this stage in spite of all treatment, in these two cases the disease was mild and only partially interfered with locomotion before plaster immobilization. After removal of the patient from the plaster cast it was found that the left hip and knee had lost all movement. Rightly, Scott calls these cases "tragic results". The performance of the Mantoux test might have prevented some of these tragedies. If we follow the injunction of Scott, fewer living immobilized statues may be seen eking out their miserable lives hoping for the relief of death:

> To avoid serious errors in diagnosis, and the tragic after-results, no case of suspected tubercular disease of the hip joint should be immobilized until after a careful radiological examination of the sacro-iliac joints.

Calcification and Ossification of Vertebral Ligaments in the Absence of Other Vertebral Lesions.—In many cases the condition is discovered accidentally and there are no symptoms. Oppenheimer[15] stated that of 18 patients in this group, 17 were aged over fifty-five years.

The condition is most common in the thoracic area and uncommon in the cervical area, an area of great mobility. Oppenheimer states that calcification occurs on the anterior or ventral surface, where the tension and mobility are least. Slight rarefaction is present. He concludes that "lime salts liberated by rarefaction of vertebral bone may be deposited in the vertebral ligaments, the tension or mobility of which is diminished". In his opinion "the findings seem to indicate that two factors are common to the different forms of calcification and ossification in the vertebral ligaments, namely, (i) disease of ligaments, (ii) rarefaction of the vertebral bones". This type of lesion provides compensation courts with some of their contentious cases, especially when some slight blow received months earlier is blamed for the condition. Whether this type of disorder, by occurring at a later age, is really an attenuated type of ankylosing spondylitis is a moot question. It should be borne in mind that young men may be found with "poker backs" which apparently have been almost symptomless, yet the radiological evidence warrants the diagnosis of true ankylosing spondylitis, and the subsequent course of the disease has corroborated this opinion.

Treatment.

The treatment of ankylosing spondylitis has undergone a change during the last ten years, as X-ray therapy and orthopædic support became established as remedies. There is still a great deal of ignorance, not only regarding the disease, but also as to the efficacy or otherwise of X-ray therapy.

Before the advent of this latter form of treatment, many measures had been tried. Drugs, vaccines, gold, fever therapy, and prolonged orthopædic treatment—all had their day, and very little of any of them except orthopædic treatment has remained. Vitamins and various forms of physical therapy were all tried, but none gave much hope of relief. The disease in some cases reached a standstill and the patients carried on for years; but in other cases slowly progressive disease or some intercurrent infection was the patient's misfortune. F. C. Hall,[17] in a review of various methods of treatment in 1943, stated that gold treatment was not effective, X-ray therapy was considered effective by some, and plaster and leather jackets were often used effectively. Tyson found gold, fever and vaccine therapy disappointing, and Hartfall and Garland, in reporting a series of eighteen cases, stated that none of the patients was cured, but five were relieved. A review of the literature reveals that two great measures—namely, X-ray therapy and orthopædic treatment—seem to have given the best results so far.

Jacques Forestier[9] in 1939 gave the following as his method of treatment: (i) rest, a jacket support for the back, bath therapy (temperature raised), massage and general physical methods; (ii) chrysotherapy; maximum dose 10 centigrammes per week, one gramme for each series, repeat in two or three months; (iii) injection of a radio-active substance. Forestier first employed thorium (50, 150 and 250 microgrammes at intervals of one week for eight to ten doses, a total of 800 to 1,200 microgrammes being given). Later he employed injections of gaseous radon, which was given with a syringe of 20 cubic centimetres' capacity, the dose being 6,000 to 12,000 millimicrocuries. The dose was given every second day, a total of 12 to 20 injections being employed. The course could be repeated in four to six months. The results were satisfactory; of 25 patients treated in the pre-ankylosing stages, 80% became free of all symptoms after one or two years, and 15% have had some relapse.

In view, however, of the pathological pattern of the disease, general measures should not be entirely discredited, and therapy found in the past to be helpful should not be thrust overboard before the revaluation of the newer methods can take place.

Before we consider the main basis of the most modern treatment of ankylosing spondylitis, it may be as well to discuss some phases of therapy necessitated by knowledge of the general pattern of the disease, and at the same time some of our own methods, past and present, will be discussed.

Diet.

The loss of weight, which is often pronounced, calls for increased diet—proteins, fats and carbohydrates. The disturbance in the plasma proteins should be treated by adequate amounts of easily digested protein, such as the dairy products and eggs. Recently one of us has given in addition 30 grammes of amino-acids per day (made from gelatin, casein and yeast by enzymatic hydrolysis, 2% of cystine being added). In addition, two grammes of cystine have been given by capsule. It has been shown that if hydrolysed protein is added to ordinary undigested protein, a more rapid and greater utilization of the latter is obtained. The cystine was added because it is the essential amino-acid in the formation of plasma proteins and also plays a role in tissue oxidation. Two teaspoonfuls

of the foregoing compound are taken three times a day in a hot "Bovril" drink, to which is added one teaspoonful of tomato sauce. This makes a palatable drink. Since Canon has shown that glycine given by mouth can enter the euglobulin fraction of the serum which is essential to our immunological defence mechanism, the hydrolysed gelatin supplies this amino-acid in abundant quantities. Green vegetables and plenty of fruit supply necessary vitamins in the diet.

Vitamins.

Exposure of the whole body to ultra-violet light seemed to benefit many patients previous to the advent of X-ray therapy. This suggested that vitamin *D* might be of value, and Buckley has laid stress on the value in this disease of cod-liver oil for its vitamin *A* and *D* content. It seems to be more beneficial than any of the synthetic combinations of vitamins *A* and *D*, which are in common use today. We have never seen any pronounced benefit from any of the other vitamins, even when they are used in large doses.

Vaccines.

Vaccines are slowly slipping backwards in popularity. In two cases in our series they were harmful and produced severe reactions, and we have stopped their use in most cases of this disease.

Medicines.

General tonics to combat anæmia and hydrochloric acid in achlorhydria are valuable. Some authorities like arsenic and others the glycerophosphates. They all can be used and varied. Sedatives are valuable generally, phenobarbitone being sufficient in most cases. Relief of pain and stiffness can generally be obtained by such substances as aspirin, phenacetin, salicylates, chloral hydrate and, if necessary, codeine, either added to an aspirin, phenacetin and caffeine mixture, or as one of the proprietary lines. In a few cases iodolysin has proved to be of distinct benefit.

Decalcification.

Decalcification suggests the intramuscular or intravenous administration of calcium, and in some severe cases associated with pronounced muscular irritability the exhibition of 10 mils of calcium gluconate once or twice a week has been valuable in reducing pain and spasms.

Chrysotherapy.

Chrysotherapy has been valueless, even failing to alter appreciably the course of those cases in which rheumatoid arthritis was present as well as ankylosing spondylitis. Affected fingers, wrists, knees and ankles have not responded, except in one case, in which "Myocrisin" (aqueous solution) was injected once a week into the knee joints, which were much swollen and evidenced a well-established polyposis. After six injections into each knee joint this man could walk without sticks, even though he had had to have their support for about one year previous to the treatment. His general condition also improved considerably.

Colloidal Sulphur.

Colloidal sulphur given intravenously or intramuscularly proved to be valueless.

Transfusions.

Transfusions of blood or plasma may be helpful in severe cases.

Rest.

The loss of weight, the raised blood sedimentation rate and the reduction of plasma albumin content all indicate the profound alteration in the body generally. If these are not severe, the patient can carry on his occupation, avoiding strenuous exercise. The latter undoubtedly aggravates the disease, and frequently the athletic patient will keep persisting in his usual exercises, trying to keep physically fit. Mild exercise is beneficial and keeps the mind away from the spectre of crippledom, which in the past has always hovered in the distance. In a number of cases one of us has had to advise patients to stop work and rest at home for many months.

An occupation demanding a lot of lifting and back-bending will aggravate the acute stage of the disease, especially if pronounced thoracic involvement is present, with its consequent limitation of chest expansion. Economic insecurity often forces the patients to try to carry on, when rest may be valuable to tip the scales towards recovery. Many of these patients are just as ill as those suffering from tuberculosis, as is revealed by general tests, and it is unscientific and inhuman to force many of them to carry the burden of disease and economic security. A temporary and adequate pension system would be helpful during the period of enforced rest.

On the other hand, complete rest in the recumbent position for months at a stretch should be avoided if possible. Sometimes the acute nature of the disease forces the issue, but we have made it a rule not to keep patients in bed longer than is absolutely necessary. In the acute cases of great severity, blood transfusions (or, better still, plasma transfusions) should be utilized to blunt the edge of the attack and enable mobility to be resumed, even though only on a small scale.

Orthopædic Measures.

Rheumatologists owe a great deal to the American orthopædists for their persistent efforts to relieve and prevent deformity in this disease. Swaim[138] carried out painstaking investigation and treatment, and reported a series of 106 patients treated over a period of twenty-two years. The group comprised 22 females and 84 males. The sacro-iliac joints fused in every case, and he states that after fusion the disease stopped. This has not been our experience. In our series one patient (Case XXXI) had a bilateral fusion operation, but the disease began to attack his thoracic area a few years later and continued to increase in severity until it was controlled by X-ray therapy. Swaim's theory was that "ossification represents the result of nature's method of permanent immobilization of the spine to prevent strain motion and inflammation in the ligaments". He used a waxed leather jacket, steeled in the back and laced in front; it was cut low in the back and high in the front, to correct the thoracic deformity. He came to the following conclusions. (i) Relief from pain, muscle spasm and strain was rapid and effective in 59 cases in which patients cooperated. (ii) The usual deformities in posture were prevented in the great majority of cases. (iii) Better health resulted, as was shown by increased weight, and ability to return to work was noticeable. (iv) Chest expansion increased. (v) Not one new hip involvement occurred in the 62 cases in which the jackets were worn, and the condition of seven out of eleven hips with symptoms of involvement improved after the jackets were applied. (vi) The experiment suggests that ossification of the intervertebral ligaments is less rapid and extensive when the spine is supported with jackets early in the disease. (vii) The results following the use of the jackets were superior to those obtained among the 45 patients previously treated with other forms of support. Some of the jackets were worn for as long as six years, others for less than one year.

We have found the Taylor brace a suitable and light support, and recommend its use when required. Patients with the condition in a mild form do not need the brace; but those with severe pain, and those in whom the disease is active and progressive, should be instructed to wear one. All patients speak of the support the brace gives and of the reduction of pain which occurs whilst it is worn during the day.

In very acute cases in which the hip joint is involved, orthopædic measures and complete rest in bed may be necessary. Vigorous medical treatment must be undertaken to get the patient back on his feet as soon as possible, as ankylosis is prone to occur. It is in these acute and progressive cases that the cooperation of rheu-

matologist and orthopædist will pay handsome dividends in relief of pain, reduction of deformity and promotion of recovery.

On no account in ankylosing spondylitis should a back or hip be immobilized in a plaster cast. If the diagnosis between this disease and tuberculosis is in doubt, it is wiser to wait for positive evidence of the latter before immobilization. Scott's statement concerning cases in which a wrong diagnosis of tuberculosis of the hip was made, which we can confirm, is as follows: "Immobilization in plaster led to disastrous results, in some cases one hip, in others both hips, became ankylosed, in addition to the stiffness of the spine."

X-Ray Therapy.

It was Kalmeter, of Stockholm, who directed our attention to the effect of X rays on the chronic rheumatic diseases, but it was Gilbert Scott who focused the spotlight of this therapy on ankylosing spondylitis.

One of us saw the first patient treated in 1931 at the Peto Place Red Cross Rheumatic Clinic; but it was not till years later that the therapy was placed on a firm basis. In his book, "Adolescent Spondylitis", Scott unfolds the picture of the disease and the wide-field therapy which he has developed in its treatment. This therapy has entirely changed the outlook in this disease from one of almost complete hopelessness to one of buoyant satisfaction with a successful outcome. It has armed the profession with a remedy able to produce more than a modicum of successful results, and therefore should help to terminate the present medical apathy and helplessness in this disease. The therapy has passed from the experimental stage and now rests on the foundation of solid results, is corroborated by some years of observation. It will not give universally successful results; it will not cure all patients or restore to normal the "poker back", the kyphotic chest or the ankylosed hip; but in a large number of cases the disease becomes quiescent and apparently cured. The therapy and some variations have been tried in a number of countries, and the reports are almost all partially or completely confirmatory of Scott's claims.

Wide-field X-ray therapy is different from deep X-ray therapy. Scott subjects the whole trunk to irradiation from one direction at each treatment. The combination of factors he uses is as follows: 130 kilovolts, 5 milliamperes, 3 millimetres of aluminium as filter, the dose being 60*r* to 100*r*. The distance between the tube and the patient is 17 to 20 inches.

The average length of the trunk is 22 inches, so that if only a 12-inch field is possible at the distance of 20 inches, then two overlapping fields are used. Scott was finally using a wide-angle tube covering the whole trunk at a distance of 17 inches. He states that the optimum constitutional effect is produced when the distance is 17 to 20 inches and the voltage not higher than 130 kilovolts, and a filter of three millimetres of aluminium has been found most suitable.

In contrasting deep and wide-field X-ray therapy Scott states that the former operates as "the larger the dose, the better the result", while the latter requires small doses at correctly spaced intervals. He remarks that "considerable experience is required in gauging not only the actual dose to be administered each session, but also the correct frequency of the irradiation. All the attributes of a physician are therefore necessary to the radiologist who gives the treatment". He tries to produce a state of saturation and keep the patient at this point. The "saturation" level is gauged by certain post-radiation phenomena, such as slight abdominal discomfort or even nausea within twenty-four hours of treatment, or alternatively, fatigue, headache, and a peculiar alteration in taste.

Some patients are sensitive even to small doses, as low as 20*r*, and the interval between doses must be reduced to two or three weeks. In our own series a number of patients reached saturation when 48*r* had been given, and in a few cases 100*r* could be administered without nausea or other symptoms.

Treatment once or possibly twice a week is usually required to keep the patient in a state of saturation. An interval of two or three weeks is allowed after ten or twelve irradiations. Over-dosage can usually be detected by weight loss, nausea *et cetera* and fatigue. Progress can be measured by gradual increase in weight, reduction of pain and stiffness, increased mobility, reduction of anæmia, and a gradual return of the blood sedimentation rate to normal.

We have followed Scott's earlier technique, using the following factors: 130 kilovolts, five milliamperes, three millimetres of aluminium filter, 17 inches focal skin distance. Two fields twelve inches square are irradiated, on the trunk dorsal area, the dosage being 30*r* to 100*r* to each field.

If the neck is affected two millimetres of aluminium and 0·25 millimetre of copper have been employed as filters, the kilovoltage has been 140, and the dosage 60*r* to 100*r*. A total of eight doses was given. Treatment took place once or twice a week according to reactions, and a period of three to six weeks was allowed between courses.

In general, patients with the condition in a severe form do not stand large dosage, and frequently only 30*r* could be given to each field every second week. Undoubtedly these patients are very sensitive to X-ray therapy.

In spite of Scott's insistence on the necessity for a low peak kilovoltage (130) in wide-field therapy, other investigators have used different techniques.

The following investigators have published complete or incomplete technical details of their methods, and their results: Weinberg,[19] Smyth *et alii*,[20] Tengbergen and Dekkers,[21] Baker,[22] Blair,[23] Hare,[24] Hilton,[25] and Raff.[26]

The peak kilovoltage will be seen to vary from 100 to 200. The milliamperage is as low as 4 and as high as 20, but is often not recorded. The filters are generally light, varying from one to three millimetres of aluminium, but in some cases a filter of one millimetre of aluminium and 0·25 to 0·5 millimetre of copper is favoured. Most focal skin distances are 50 centimetres.

Fields vary in area from 100 to 300 square centimetres, some workers using wide fields, others using narrow fields, whilst one (Hare) used six portals of entry.

Dosage varied from 60*r* to 200*r* at each application to every field; the total amount given was between 450*r* and 1,000*r* to each area, 600*r* being the most common.

Gwen Hilton used narrow fields—not the usual field of Gilbert Scott (12 inches by 12 inches), as she thought this reduced the actual dose received and diminished nausea as well as other reactions.

Therefore different techniques and dosages are used by various authors, but all claim considerable improvement in approximately 80% of cases.

Ill Effects.—Smyth[20] noticed leucopenia in three of his cases, but recovery took place in all within one month, even though the lowest leucocyte count was 1,100 per cubic millimetre. Anorexia, nausea and vomiting have occurred in many cases as reported by many authors, and suggest a narrowing of the field. We have done this for a few patients who were extremely sensitive, with a reduction of anorexia. This latter effect probably accounts for the weight loss observed in some cases, and it has been our rule to suspend therapy until the lost weight was regained. The nausea and vomiting are not altered by vitamin *B*, liver, glucose or calcium. However, in view of the beneficial effects of wide-field X-ray therapy, the ill effects are infinitesimal.

Comments of Later Investigators.—Smyth[27] states that "we consider roentgen therapy the best method of controlling the symptoms of rheumatoid arthritis of the spine. Whether the benefit is entirely symptomatic or whether the course of the disease is favourably affected will need to be determined by further investigation." Comrie states that "roentgen therapy is the best single agent in rheumatoid arthritis of the spine that they had". Gwen Hilton states that "the treatment is worth while in ankylosing spondylitis, whatever the stage, except when there is complete ankylosis coupled with entire absence of pain".

The Mode of Action.—The mode of action of X-ray therapy is unknown, but it has been suggested that it stimulates the ductless glands or increases the circulation through action on the vegetative nervous system. Blair[20] surmises that the X rays may liberate sulphur within the body by acting on the mast cells of Ehrlich, which contain a polysulphuric ester.

The blood sedimentation rate improves as the patient's condition improves, but it may rise even though no clinical improvement is obvious.

Results of Other Investigators.—Scott recorded 432 cases, with pronounced relief and benefit in approximately 80%. Six other authors,[12][13][20][31][34][35] report 326 cases with approximately the same results. They all stress the apparent complete recovery in the early cases; but radiographic evidence still remains, and ankylosis and calcification may not only be unaltered, but may increase. Gain of weight, cessation of pain and stiffness and a normal blood sedimentation rate are better criteria of successful therapy than radiological evidence, although sclerotic changes in the sacro-iliac joints point to good defensive mechanism or cure. The return of the plasma proteins and the results of liver function tests to normal should give confirmatory evidence.

The Results in the Present Series.

The following are some observations on a number of our patients who have been treated by X-ray therapy. This series of 38 patients treated since 1938 has been under observation from one to five years; 10 patients have been observed for between four and five years, and the rest for varying periods up to one year. There were 33 males and five females, showing the remarkable preponderance of one sex over the other.

In 32 estimations of the blood sedimentation rate made in the initial stages, 22 gave figures above 12 millimetres at the end of one hour (Westgren—clear column). The highest figure was 92 millimetres, and strangely, this patient carried on active work in a shop and consulted us for œdema of his lower limbs. He gave a history of Neisserian infection twenty years previously, and of recurring attacks of lumbago, not severe enough to confine him to bed. Radiologically the disease was widespread through the whole spine. In Case VIII the condition was active and acute, having produced reduction of the chest expansion to three-quarters of an inch; yet the X-ray film revealed an early sclerosis of the left sacro-iliac joint, and the blood sedimentation rate was only 24 millimetres. In Case I the patient was a male, aged twenty-six years, and the disease was extremely active, having begun at Milne Bay in the wet season and rapidly involved the whole spine. There was considerable decalcification of the whole spine and the blood sedimentation rate was 40 millimetres at 60 minutes; but the hippuric acid test produced the result of 113%. The patient had lost two and a half stone in weight. In Case IX the patient was a male, aged twenty-two years, who had been placed in a plaster cast for supposed tuberculous disease of the hip. He later developed bilateral sacro-iliitis and a "poker back", his chest expansion was considerably reduced, and his blood sedimentation rate was 53 millimetres at the end of one hour. He was fortunate in not having any ankylosis of his hip joint after immobilization in a plaster cast; but the latter did not prevent the disease from advancing and later involving the thoracic part of the spine. In Case XXVII the disease was clinically and radiographically active in a young man whose blood sedimentation rate was six millimetres in the first hour.

We have found that the blood sedimentation rate does not in all cases indicate the severity or the progressive nature of the disease. This is important, and is stressed because these two features of the disease can be assessed only by the symptoms of pain, stiffness and loss of weight and by the signs of gradually increasing reduction of mobility and the results of serial radiographic examinations.

The hippuric acid test of liver function has been and is being performed, to see whether it may throw some light on the quiescence or activity of the disease. Table I, based on a number of cases, shows that this test does not parallel the blood sedimentation rate test.

TABLE I.

Case Number.	Blood Sedimentation Rate. (Millimetres in the First Hour.)	Hippuric Acid Excretion. (Percentage of Normal.)
I ..	40	113
II ..	20	76
III ..	13	113
VIII ..	24	111
IX ..	53	112
X ..	25	62
XXII ..	13	120
XXV ..	27 65	65 90
XXX ..	3	105
XXXVIII ..	32 6	71 117

Plasma protein estimations are now being carried out, to check them against the blood sedimentation rate and the hippuric acid excretion test.

Repetition of the blood sedimentation rate tests is valuable in attempts to assess when recovery is taking place, especially when it is realized that the X-ray appearance of increased ligamentous calcification may denote either progression of the disease or recovery and healing.

In Case XXVIII the following blood sedimentation rates were obtained over two and a half years: 22, 21, 15 and 5 millimetres. These figures coincided with clinical improvement. In Case XXXVI progressive figures over a period of two years were as follows: 63, 42, 35, 22 millimetres. After he recommenced work the patient's condition remained the same, but eighteen months later recurrence of the pain and stiffness took place, and the blood sedimentation rate rose to 55 millimetres. We have concluded that the sedimentation rate and the clinical condition together reveal the activity of and progress of the disease more than either separately.

Achlorhydria did not occur in any greater numbers of cases in this series than in rheumatoid arthritis.

Anæmia and the "shift to the left" of the neutrophile leucocytes are not common and often do not parallel the blood sedimentation rate or the clinical condition. In one case the anæmia increased under X-ray therapy, even though the dosage was small (30*r* at each treatment).

The fact that the blood sedimentation rate may not parallel the clinical improvement has been noted by a number of authors.[35][20] In Case X the patient suffered from nausea and was given a blood transfusion, the original blood sedimentation rate (25 millimetres) increased to 55 millimetres and later to 60 millimetres, and her condition was unsatisfactory even after one year. A relapse is generally accompanied by an increase in the blood sedimentation rate, especially if treatment has not been given for many months, as well as by a return of pain, stiffness and loss of weight.

It has been and will continue to be our practice to check the patient's condition clinically and by the blood sedimentation rate each year after the therapy has finished, if it is possible. This is the only way to control a disease which shows a great capacity to relapse, even after clinical and other tests reveal a return to normality. Regular weighing of the patient is important; it will reveal either overdosage of X rays or an increase in the disease.

At the commencement of our use of X-ray therapy we used rather large doses at each sitting—60*r* to 100*r*, generally twice a week; but we were quickly forced to use the smaller dosage once a week, because of nausea, loss of weight and appetite.

It is our practice to begin therapy as follows. The factors are as follows: 130 kilovolts peak, five milliamperes, three millimetres of aluminium as filter, focal skin distance 17 inches. The dosage given is 30*r* to two fields measuring

12 inches by 12 inches. The dosage is increased to 48*r* and then to 60*r* and given once a week, rarely twice a week, for 10 to 12 treatments according to reactions. Nausea is common and may occur in patients whose blood sedimentation rate is low or high.

Latterly we have reduced our fields to a narrow field, 12 inches long and 5 inches wide, for patients suffering from nausea, and have succeeded in reducing the latter somewhat. If progressive weight loss occurs, therapy is stopped for two or three weeks and the weight is rechecked, and if the loss continues in spite of absence of nausea, therapy is recommenced. In our opinion vitamin *B* and glucose or parenteral liver injections do not alter or prevent nausea. Nausea calls for reduction of dosage and area besides extension of time between treatments.

Case X proved to be difficult, and nausea, loss of appetite and anæmia caused temporary suspension of therapy. The patient continued to react to a dose of 30*r* given over the lower dorsal vertebræ, although a dose of 80*r* was given to her neck without the production of nausea and with considerable improvement in that area.

It has been our practice to give the second course of treatment after an interval of three to six weeks, and if symptoms have disappeared, to recheck the patient's clinical condition in three months and test the blood sedimentation rate. If the disease is active, as evidenced by one or the other test, another course of treatment is given, and after an interval of six weeks the blood sedimentation rate is again estimated. Further courses of treatment depend on clinical evidence and on the blood sedimentation rate. In some cases an interval of two years has been followed by a recurrence, and therapy has again been instituted, bringing about cessation of pain and stiffness. In one case therapy has continued for two and a half years, and during the last two years two courses have been given each year. The patient's anæmia and all pain and stiffness have completely disappeared. The therapy has not altered his leucocyte count. We have not seen leucopenia following such small dosages, and in only one case did increase in the anæmia accompany the disease.

Comment.

The results are by far in advance of those obtained by all other methods and we can add to the record of other investigators. It is approximately six years since X-ray therapy was commenced and we can record a greater success and confidence than with any other method.

In place of the depressed feeling of therapeutic ineptitude and helplessness which we formerly had, we now feel that we have a remedy which, with care and watchfulness, besides a measure of tenacity and purpose, will control the condition in about 80% of cases. It demands an extended time of treatment—often years—and the cooperation of the patient; but these are a small price to pay for a degree of therapeutic effectiveness and efficiency previously unknown in the armamentarium of the rheumatologist engaged in treating this most stubborn of diseases.

Of 38 cases, treatment has been unsatisfactory in two; in one the lesions has progressed to crippledom, the other patient is still under treatment. Four patients had relapses but have again responded to treatment, 32 have shown improvement, in some cases amounting to apparent cure and in the others to pronounced improvement in weight, blood sedimentation rate, stiffness and pain.

The X-ray evidence of the result is difficult to assess, because some patients who are clinically well, with normal blood sedimentation rates, are found to have increased calcification of their ligaments, even in the absence of pain or stiffness.

In a number of cases a considerable increase of movements has occurred in back flexion, chest expansion and neck mobility. The outlook of the patients under successful X-ray therapy is the most important guide to the effect of this therapy on a disease which, until the advent of this form of treatment, was the shuttlecock of clinician and orthopædist, and the happy hunting ground of the "quacks" known or unknown by their various labels.

Because this disease attacks in many instances the healthy, athletic, young adult male, producing financial and social loss to the community, it demands closer investigation, more careful diagnosis and the inauguration of a therapeutic regimen which has proved its great efficacy over the last ten years, instead of a palsied inactivity and a therapeutic vacancy which have characterized the general medical attitude in the past.

If a young man can recommence work after an absence of three years, during which period he was receiving an invalid pension, the use of X-ray therapy is justified and indicated. This occurred in Case XXXVI.

One important point must be stressed, and that is persistence on the part of the doctor and patient. Some patients show rapid improvement after the first course of treatment, even if the total dosage is less than 600*r*, but others only after two, three or even four courses. However, even in these latter cases some measure of relief is generally apparent after one or two courses and provides the necessary incentive for the patient to continue.

The Future.

The therapeutic effectiveness of X-ray therapy can best be ascertained in clinics set apart for the investigation and treatment of rheumatic diseases as well as providing for the inevitable follow-up, enabling time, the most critical of all judges and the wrecker of false premises and disorientated results, to deliver its final verdict. Where are such clinics?

Summary.

1. *Spondylitis ankylopoietica* and its synonyms are defined and discussed. The need for a common international nomenclature is stressed.

2. The history of the disease reveals that it is not a product of modern civilization.

3. Most writers, in discussing the pathology and histology, stress the similarity between the microscopic changes in this disease and those found in rheumatoid arthritis. Vertebral decalcification and ligamentous decalcification are prominent features, but no histological examination of the sacro-iliac joints in the early stages has been possible.

4. The ætiology still remains obscure. The common association of tuberculosis and the antecedent history of gonorrhœa are probably important and are widely publicized by the European authorities, but as factors in causation receive little support from English investigators. The role of the streptococcus in this disease is undetermined, and the recognition of a chronic brucellosal type of ankylosing spondylitis further complicates the ætiology.

5. The greater incidence in males than in females, the frequent occurrence of the disease in puberty and early adult life, and its tendency to attack healthy young athletes are discussed, mention being made of the resultant economic loss.

6. Climate does not influence the disease.

7. Under the headings mode of onset, symptoms and cause, the important prespondylitic symptoms are emphasized, and the cause of the disease is traced from the mild to the severe variety. The dictum of the British Red Cross Society and the Charterhouse Clinics is invaluable, namely, "the sacro-iliac joints are to be examined radiographically in all patients between the ages of fourteen and twenty-five years complaining of the indefinite prespondylitic symptoms". The importance of sciatica as a forerunner of the disease is noted. Rigidity, root pains and deformity are the final picture. The occurrence of iritis in non-gonorrhœal cases is worthy of interest.

8. The thyreoid gland may be enlarged and toxic, but the parathyreoids are affected only by compensatory hypertrophy.

9. A slight degree of secondary anæmia and an increase of the white cell count are often observed. The blood sedimentation rate is raised in acute cases, but may be normal in slowly progressive disease. The hippuric acid excretion

test often produces low figures, but the result is not always altered proportionately to the blood sedimentation rate. Hypoalbuminuria and hyperglobinæmia are commonly seen. A disturbance of the secretion of hydrochloric acid has been noted in about 50% of cases.

10. The difficulties of diagnosis in the pre-spondylitic stage are stressed, and the ease of recognition in the stage of rigidity and deformity is in contrast with the former. The skiagram of the sacro-iliac joints gives the diagnostic cue.

11. Most observers point to the sacro-iliac joint as the first joint involved, whilst others stress the early involvement of the apophyseal joints. Three stages in the radiographic picture of the sacro-iliac joints are recognized by both Gilbert Scott and Forestier. Some emphasis has been laid on the oblique view of the spine to reveal ligamentous calcification.

12. *Spondylitis osteoarthritica* is easily differentiated by the radiographic appearances. Malignant disease of the spine in its earlier stages, when the radiological appearances are negative, presents difficulties; but the history, response to short-wave diathermy and time may be necessary to make a diagnosis. Tuberculosis may be suspected in the sacro-iliac joints or in the hip joint; but a carefully taken history, the X-ray appearances of the lungs and the Mantoux test should enable this mistake to be avoided. The danger of plaster immobilization in ankylosing spondylitis is firmly stressed.

13. In treatment, gold, protein shock and vaccines have been tried without much success. Good and plentiful food to prevent weight loss, besides tonics and vitamins *A* and *D* or cod liver oil, is useful. The most useful methods of treatment agreed to by most authorities today are orthopædic support and X-ray therapy. The opinions of many of the authorities dealing with those two varieties of therapy are given. Forestier used thorium and later radon implantations along the spine. Transfusions of blood and plasma in suitable cases have been found to be valuable, and later the amino-acids may be of advantage. Calcium given intramuscularly or intravenously—10 mils of a 10% solution of calcium gluconate—has proved helpful in reducing the muscle spasms which are so common. Under the heading of X-ray therapy mention has been made of the wide-field therapy of Gilbert Scott, an intermediate peak kilovoltage and the larger peak kilovoltage and the larger dosage of other workers being used. Nearly all workers report 80% of good results. During five years we have treated 38 patients by wide-field therapy, using 130 kilovolts peak with two aluminium filters and small dosage (30*r* to 60*r*) at intervals of a week. The total number of treatments is 10 or 12, according to reactions, and up to three courses have been given in one year. The need for persistence of the patient and doctor is stressed. Our results are similar to those of other clinicians. The future is discussed, as well as the great need for suitable clinics.

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TREATMENT OF GUNSHOT WOUNDS OF THE CHEST IN THE FIELD AIDED BY PENICILLIN THERAPY.

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A DISCUSSION of 32 cases of gunshot wounds of the chest treated during a recent campaign in the South-West Pacific Area is presented. Wounds of the chest wall alone and abdomino-thoracic wounds are excluded. The missiles included rifle and machine-gun bullets, and grenade, mortar and high-explosive shell fragments.

Chest Wall Lesions.—The chest wall wounds varied from simple perforations with minimal soft tissue and bone destruction (15 cases) to wounds requiring surgical excision (17 cases). Many of the latter wounds were of considerable magnitude, and some were sucking wounds (seven cases). Rib fractures, involving one to four ribs, were present in 15 cases; in six of these accompanying fractures of the sternum, scapula or humerus were also present. The humerus alone was fractured twice and the scapula once. Concussion nerve lesions were present in three cases—a complete brachial palsy in one and a radial palsy in two. Many of the wounds were contaminated by dirt and clothing, but none were clinically infected when first examined.

Intrathoracic Lesions.—The thoracic contents were damaged in three ways. Firstly, the missile entered the thoracic cavity (23 cases). Secondly, when ribs were struck tangentially, comminuted bone fragments were sprayed into the thoracic cavity (four cases). Thirdly, the missile caused subpleural injury by its concussive effect as it passed close to the thoracic cage without damaging the ribs or penetrating the pleura (five cases).[4]

Laceration of the lung with blood extravasation into its tissues is necessarily present to a greater or less degree in all instances of lung injury. It was seen on direct inspection of the lung shortly after receipt of the wound in five cases, but in only 14 was extravasation pronounced enough to be diagnosed radiologically. At times it was widespread, the whole of one lung and the upper lobes of both lungs being involved in two instances each. Only once was this condition due to coincidental lung blast.

Metallic foreign bodies, often multiple and associated with rib fragments, entered the lung in 24 cases and were retained in 12, both lungs being affected in one case. None of the foreign bodies were of large size, but they caused a broncho-pleural fistula in three instances. Large foreign bodies were found in the pleural cavity twice.

Blood was present in the pleural cavity in 25 cases, in 17 of which it was accompanied by air. Massive hæmothorax was present four times, but in only one case did it cause serious signs of increased pleural tension (Case VI).

Three patients had a simple pneumothorax and three a tension pneumothorax (Cases IV, IX and XI). One of these patients (Case IX) died shortly after admission to hospital, and the other two developed a hæmopneumothorax.

The mediastinum was directly injured in four instances by the missile, which actually traversed it in Cases VI and X and exposed it by fracturing the sternum in Cases I and III. In the former three cases the missile also injured both lungs. The heart and great vessels were not involved, though in Case III a foreign body was embedded in the fibrous pericardium.

Mediastinal emphysema, characterized by friction sounds of pericardial type in the sternal and pericardial regions, occurred in four cases. In Cases I and VI air was introduced directly by the missile, in Case VIII it tracked down from a cervical perforation of the trachea, and in Case IX it spread from the lung. In the last-mentioned case, the foreign body caused an interstitial lung emphysema from which air tracked through the hilum to the mediastinum and thence into the neck and subcutaneous tissues. A tension pneumothorax was also present, probably owing to a minute rupture of the mediastinal pleura,[5] though certainly at autopsy no leak of air could be demonstrated from the lung or mediastinum into the pleural cavity. (This was the only instance of tension pneumothorax in which evidence of mediastinal emphysema was present. In Case IV it was due to a broncho-pleural fistula, and in Case XI it was probably due to escape of air from the lung along the missile track.) Air in the mediastinum may also track into the lungs, as in Case VI. The air spread into the neck in all these cases, but only once was the whole body affected.

Treatment.

The tactical situation was such that casualties were in most instances able to reach the surgeon within two to twelve hours of being wounded. In the early stages of the campaign, the casualties were admitted direct to a field hospital where full surgical, nursing and X-ray facilities were available. As the lines of evacuation lengthened, initial surgical treatment was performed in a forward unit, where again operating and nursing facilities were excellent. Refrigeration was always available for the storage of blood and penicillin. There were no X-ray facilities here, but as most patients were able to be sent to the field hospital in a few days, little inconvenience was caused by their absence, clinical signs being generally sufficient for the early management of these patients. The wounded were held here until they were fit to undertake a short sea trip back to the field hospital; this was usually from five to ten days after receipt of the wound, according to the severity of the lesion.

First-Aid Treatment.

By the regimental medical officer at his aid post morphine was given and sucking wounds were closed by deep silkworm sutures. Each patient was watched for signs of raised intrapleural tension during his evacuation to the surgeon.

Treatment at the Operating Unit.

Tetanus toxoid and gas gangrene antiserum were given as a routine measure, and intramuscular penicillin therapy (15,000 units every three hours) was commenced.

Disturbances of lung physiology due to increased intrapleural tension took precedence in treatment. Tension pneumothorax was relieved by an indwelling needle. Massive hæmothorax was immediately aspirated if it was causing distress. Oxygen was administered when necessary. Morphine and rest were sufficient to relieve the "thoracic shock"[3] common in these cases; but in many instances the shock-like state was due to blood loss, which necessitated blood transfusions for resuscitation.

When resuscitation was completed, the patient was radiologically examined, if facilities were available, and taken to the operating theatre when surgical measures were necessary for wound treatment. (Small, clean, perforating or penetrating wounds did not require surgical excision.)

Satisfactory anæsthesia was obtained by the intravenous injection of "Pentothal". This was supplemented by oxygen given through a close-fitting facepiece. By means of an adjustable valve the lung was able to be deflated or inflated as required—as, for instance, when a large sucking wound was being treated.

Any sutures already present in the chest wall wound were removed and the wound was then excised in the usual manner. If the pleural wound was small, it was not interfered with and no effort was made to inspect the pleural cavity or lung. When the wound was larger, but there was no indication for further exploration, the only intrapleural manipulation performed was the sucking of the blood from the thoracic cavity. When the pleural wound was large and the lung was widely exposed, the thoracic cavity was inspected after adequate retraction had been obtained with rib retractors. In such cases the ribs had usually been so damaged that further resection or division was unnecessary. All blood and foreign bodies were evacuated from the pleural cavity and the lung was examined. The extent of lung injury was noted and the presence of air leak was sought. Visible foreign bodies were removed. In no instance was any other manipulation necessary, as hæmorrhage had always stopped and no large foreign bodies were present in the lung. The lung was then inflated to the chest wall, and 50,000 units of penicillin were instilled into the pleural cavity. The pleura (when possible) and the overlying soft tissues were sutured. The pleural cavity was not drained, reliance being placed on aspiration to remove any post-operative collection of fluid. The chest wall tissues were then insufflated with penicillin-sulphanilamide powder. If conditions were favourable, primary suture of the skin was performed. Otherwise, the skin was left open, the wound was padded with "Vaseline" gauze, and delayed primary suture was performed four or five days later.

Thoracotomy by a separate incision had to be performed only once. In Case III a bullet, entering at the apex, was lying on the diaphragm and causing severe pain and spasms of coughing. This was easily removed through a small postero-lateral intercostal incision.

After operation watch was maintained for signs of increased intrapleural tension, as a massive hæmothorax or tension pneumothorax may quietly and unexpectedly develop with few indications until thoracic compensation suddenly breaks down.

In all cases of hæmothorax aspiration was performed as far as possible within twenty-four hours of receipt of the wound.[4][7][16] The physical signs of hæmothorax following gunshot wounds may be anomalous, especially in the presence of air,[4] so that a chest containing 500 millilitres of fluid in it may show few signs of fluid. Even X-ray examination may miss collections of fluid of less than 400 millilitres. In Case V a lack of physical signs delayed aspiration of a hæmothorax until the seventh day after receipt of the wound. Thereafter, aspiration was always performed, not only when fluid was known to be present, but in all doubtful cases as well. As much blood as possible was removed at each aspiration. In four instances over a litre of blood was slowly removed without ill effect to the patient.

Aspiration, with X-ray checks wherever possible, was repeated every twenty-four to forty-eight hours until the chest was free from fluid. In many cases the first one or two aspirations produced only an ounce or two of blood, but much larger amounts of increasingly serous fluid were withdrawn at later sittings. A two-way tap was used to prevent air from entering the pleural cavity,[117] and cultures were attempted from each specimen of fluid.

Careful watch was maintained for the presence of clotted blood in the thorax.[4][10][25][30][33] There were no instances of early true massive clotting;[30] but there were four cases of the later fibrinothorax due to clotting of the effusion poured out as a result of the presence of blood in the pleural cavity.[4][30] As soon as this condition was diagnosed thoracotomy with rib resection was performed, the clots were removed from the pleural cavity, and the fibrin and early granulation tissue were stripped off the lung and the parietal pleura.[27][32] This procedure was then followed by suction drainage. More complicated cases—for instance, those in which a broncho-pleural fistula was present—were referred to base.

When infection occurred in the pleural cavity, aspiration and penicillin instillations were continued until the pus thickened and formed an empyema and the lung failed to expand further. Thoracotomy was then performed.

Chest Exercises.—As soon as the patient was well enough, routine supervised chest exercises were commenced. This is a most important aspect of the treatment of these patients,[2][3][5][14][30] as even a simple hæmothorax may be followed by rapid immobilization and chest deformity in the absence of exercises.

Penicillin.—Penicillin was given intramuscularly in amounts of 15,000 units every three hours in all instances. Penicillin was introduced into the pleural cavity in all cases of hæmothorax; 50,000 units were instilled after the first two aspirations as a prophylaxis against infection. If infection with penicillin-sensitive organisms occurred, then 100,000 units were instilled every day after aspiration until thoracotomy was performed; 100,000 units were then instilled through the drainage tube (which was clamped for three hours afterwards) each day until no organisms could be grown on attempted culture from the discharge. This usually occurred about the fourth day, provided no broncho-pleural fistula was present.

Blood Transfusions.—Unless they are due to gross upsets in pulmonary physiology, the majority of the shock-like states seen in chest wounds (as in all war wounds) are due to loss of blood, either internally into the lung, pleura and parietes, or externally.[4] Such patients require stored or fresh blood for adequate resuscitation. In this series 11 required blood transfusion on their admission to hospital. One or two litres (in one case three litres) were the usual amounts required. Blood was never given as rapidly as in other injuries, in order that the pulmonary circulation should not be overloaded, though in two severe cases one litre was given in the first half-hour with no ill effects. Blood transfusions are just as important in the after-treatment of these patients,[22] and anyone with a hæmoglobin value below 80% was given a transfusion. (Blood hæmoglobin estimations were performed every three days.) Sixteen patients required blood for anæmia due to the original blood loss. This was accentuated by intrapleural infection in five cases. One to three litres of blood were usually sufficient to correct the anæmia, and normal blood hæmoglobin values were obtained in from one to three weeks. Three patients with pleural infection still required blood transfusions after four weeks, and they had received an average of eight litres by then.

Results.

Two patients died within a few hours of treatment, one (Case IX) of shock following an apparently successfully treated tension pneumothorax and the other (Case VIII) of shock and blood loss due to severe chest and neck injuries. Both of these had mediastinal emphysema.

Parietal Wounds.

Sixteen patients required surgical excision of the chest wall wounds. Nine wounds were completely sutured by primary suture, and in only one case did this fail, owing to gaping after removal of the sutures. This wound was successfully resutured. In the remaining seven cases skin suture was performed about the fifth day. All sutures were successful, except in Case III, in which the wound became infected locally with a penicillin-sensitive *Staphylococcus aureus* and *Bacillus proteus*. Fourteen patients did not require wound excision, and all their wounds healed by first intention.

Those who had no intrapleural infection received an average amount of 720,000 units of penicillin intramuscularly for treatment of their parietal wounds. Those with intrapleural infection received far larger amounts, as penicillin was used systemically to treat the intrapleural condition as well.

Intrapleural Lesions.

The lung expanded in from three to four weeks in cases of simple pneumothorax. When extravasation of blood into the lungs was the only lesion, the lung fields were radiologically clear by the end of five weeks at most.

Hæmothorax.

Hæmothorax is the most important and frequent complication of gunshot wounds of the chest.[2][4][22] Of the 24 patients who had blood in the pleural cavity, 18, including three with large sucking wounds, responded to early and repeated aspirations alone after initial surgical treatment, and in none did infection occur. The number of aspirations averaged four (varying from one to ten), and the chest was free from fluid in an average of twelve days (the period varying from four to twenty-one days). The total amount of fluid aspirated averaged 900 millilitres (varying from 150 to 3,000 millilitres). Each patient received an average of 100,000 units of penicillin into the pleural cavity and 720,000 units intramuscularly.

In a further case (Case V) no infection occurred, but thoracotomy was later required for the evacuation of a fibrinohæmothorax complicated by a broncho-pleural fistula.

Five hæmothoraces became infected, one by a penicillin-sensitive hæmolytic streptococcus with *Bacillus proteus*, and four by a penicillin-sensitive *Staphylococcus aureus*, in two cases associated with *Bacillus proteus*.

In Case VII a large uniloculated hæmothorax was present, which became infected by a penicillin-sensitive *Staphylococcus aureus*. Gross pus was never formed, and the condition yielded to aspiration and to the intrapleural and systemic administration of penicillin, so that the chest lesion was completely healed in three weeks. This case was not one of empyema; it was one of infected hæmothorax which penicillin prevented from becoming an empyema.[9][12][13][21]

In Case II a localized infection developed in the lung and burst into the pleural cavity, forming a small basal empyema. Penicillin sterilized the fluid but did not avert the formation of thick pus, which required a thoracotomy.

In Case VI a fibrinohæmothorax developed, which became infected; it responded well to thoracotomy and removal of the fibrin clots.

In Cases III and IV an infected fibrinohæmothorax was present, due to a persistent broncho-pleural fistula, and thoracotomy was required.

The amounts of penicillin used in these five cases may be seen in the individual case reports appended.

Fibrinohæmothorax.

The presence of blood in the pleural cavity causes an outpouring of serous fluid. Clotted hæmothorax may be due to clotting of the actual blood (usually within twenty-four hours of receipt of the wound), but more commonly it is due to later clotting of the serous effusion causing a fibrinohæmothorax.[30] The cause of this clotting is not yet fully explained, but it is known that infection is not the only factor involved, as only 30% of such hæmothoraces are infected.[31][32] The incidence of the condition in a large series appears to vary from 5%[7][9][30] to 9%.[32] In four cases (Cases III, IV, V and VI) in this series fibrinohæmothorax developed, and in three it became infected, in two owing to a broncho-pleural fistula.[6][11][18] Case V was also complicated by a broncho-pleural fistula, but no infection supervened. All these patients were treated by thoracotomy and decortication; but in only one (Case VI), in which no fistula was present, was healing complete in six weeks.

Thoracotomy.

Exploration of the pleural cavity was necessary at the initial operation in four cases, owing to the presence of large sucking wounds, and once owing to the presence of a large foreign body in the pleural cavity (Case II). The last-mentioned was the only one in which a separate incision was required. In three of these (including Case I) healing was rapid, with dry pleura and normal lung function; but in two a later thoracotomy was required in addition. In Case II a small basal empyema developed, owing to lung infection, which rapidly cleared up with drainage, and in Case III an infected fibrinohæmothorax developed owing to a broncho-pleural fistula.

Late thoracotomies were performed in Cases IV, V and VI for fibrinohæmothorax.

Retained Foreign Bodies.

Twelve patients had small retained foreign bodies in the lung. In one case only were symptoms due to this—namely, cough and hæmoptysis lasting for three weeks.[1] All these patients were able to be followed up for three months, and there was no indication for removal of the foreign body during this time.

Discussion.

The benefits of early and frequent aspiration for hæmothorax, without air replacement, are well recognized.[6][14][17][24][30] Such treatment hastens the return of normal pulmonary function by the prevention of clotting and infection of the uncomplicated hæmothoraces.[2] Should clotting occur, then thoracotomy with removal of the clot and parietal and pulmonary decortication must be performed[32] in order to prevent the onset of fibrothorax, in which the fibrin on the lung and parietal pleura is rapidly replaced by granulation tissue and later by fibrous tissue.[15][20][32] This leads to permanent thoracic crippling and chest deformity. Calcium may even become deposited in the pleura in late cases.[20]

Penicillin used as an adjuvant to surgical treatment is most valuable in chest wounds. Intramuscular penicillin therapy prevents infection in the parietal wound by penicillin-sensitive organisms and aids early suture. It prevents infection from occurring along the needle track or round thoracotomy wounds in the presence of intrapleural infection. It also prevents or localizes infection arising in the lung itself and may even sterilize effusions in the pleural cavity.[5]

Penicillin instilled into the pleural cavity remains there for twenty-four to forty-eight hours.[28][30] Used as an adjuvant to aspiration, it helps prevent infection of hæmothoraces,[29] and sterilizes and helps localize simple infected hæmothoraces or even empyemata.[8][14][18] It cannot sterilize infected fibrinohæmothoraces owing to their numerous loculi, nor as a rule can it sterilize the hæmothorax in those cases in which a broncho-pleural fistula occurs. Such a fistula constantly reinfects the pleural cavity and is one of the most difficult to treat of all chest wound complications.[19] The value of penicillin in such cases is that it localizes the infection and prevents spreading sepsis. In all cases of empyema, whether the condition is sterilized or not by penicillin, a thoracotomy must be performed as soon as the pus is too thick to aspirate and the lung has ceased to expand.[8][32] The problem here is not only to sterilize the effusion, but to aid expansion of the lung. A sterile empyema is not a cured empyema,[9][10][21][29] because fibrothorax rapidly follows undrained chronic, though sterile, empyema.

In conclusion, the mortality among patients who sustain gunshot wounds of the chest and live long enough to undergo surgical treatment, has dropped from 24% in World War I to 8% in the present war,[12][26] owing especially to improved methods of treating shock, to early surgical treatment, to early and frequent aspiration of hæmothoraces, and to the use of penicillin.

Reports of Selected Cases.

CASE I.—A submachine-gun bullet caused a large, transverse, gutter wound of both pectoral regions, stretching from one nipple line to the other. The bullet travelled along the shaft of the right second rib and through the manubrium and upper part of the sternal body, causing comminuted fractures. It then went anterior to the left second and third ribs, exposing but not fracturing them. A sucking hæmopneumothorax was present on the right side, with laceration of the upper lobe of the lung, in which were many small foreign bodies and a wide extravasation of blood. The upper lobe of the left lung was also bruised by the concussion effect of the bullet. A small portion of the anterior and upper part of the mediastinum was visible and was seen to be bruised. Mediastinal emphysema was present as well, and it had spread into the root of the neck. The wound was excised, the blood was sucked out of the pleural cavity, penicillin was instilled and primary suture was performed. The wound healed by first intention, no fluid recurred in the chest, and the lung fields were clear in three weeks. The amount of penicillin used was 100,000 units into the chest and 600,000 units intramuscularly.

CASE II.—A rifle bullet perforated the right lung from apex to base and was retained in the pleural cavity, lying free on the diaphragm. It caused severe pain and frequent attacks of coughing. Fourteen hours after receipt of the wound the bullet was removed through an intercostal incision. All blood was sucked out of the pleural cavity, penicillin was instilled and the wound was closed by primary suture. The chest was aspirated and penicillin was instilled every second day thereafter. The bullet track in the lung became infected and discharged into the pleural cavity, forming a small basal empyema from which a penicillin-sensitive *Staphylococcus aureus* was grown on culture on the sixth day. Daily aspiration and penicillin instillations were continued, and the pus was sterile on the tenth day. However, it was now thick, so the empyema was drained by rib resection. Convalescence was uneventful, and the chest was normal one month later. The amount of penicillin used was 400,000 units into the chest and 1,600,000 units intramuscularly.

CASE III.—A high-explosive shell fragment caused a large, penetrating wound of the chest, involving the sternum and the anterior costal margin on the right side, so that a sucking hæmopneumothorax occurred. Metallic foreign bodies were present in the bruised and lacerated lower lobe of the lung, the diaphragm and the pleural cavity. Two loose costal cartilages were also present in the pleural cavity, and a large piece of metal was embedded in the exposed fibrous pericardium in front of the right ventricle.

The sucking wound had been closed shortly after receipt of the wound, but the patient did not reach further surgical aid for thirty hours. The wound was excised and the foreign bodies were removed from the pericardium and the pleural cavity. The lung was seen to be lacerated, but it was not bleeding and there was no evidence of broncho-pleural fistula. After the blood had been sucked out, the thoracic cavity was closed by apposition of the deep tissues. The skin was sutured on the fifth day, but superficial wound infection by a penicillin-sensitive *Staphylococcus aureus* and *Bacillus proteus* caused it to break down. It was later successfully resutured after removal of a sequestrum from a rib.

[1] Since this paper was submitted for publication we have learned that this patient developed round the foreign body a small pulmonary abscess which was cured by removal of the foreign body five months after the patient was wounded.

In spite of aspiration and penicillin instillation every second day, a fibrinohæmothorax formed and became infected with the same organisms as the wound. Rib resection thoracotomy was performed on the eighteenth day, when a large loculated fibrinohæmothorax was found complicated by a broncho-pleural fistula. As soon as the patient was well enough he was transferred to the mainland for further treatment. The amount of penicillin used was 400,000 units intrapleurally before thoracotomy, 3,000,000 units after thoracotomy (before transfer) and 1,600,000 units intramuscularly.

CASE IV.—A rifle bullet traversed the right lung, producing a tension pneumothorax, which was followed by a hæmo-pneumothorax. There was no evidence of mediastinal emphysema. The wound did not require excision. In spite of repeated aspiration and penicillin instillations from the second day after receipt of the wound, a fibrinohæmothorax developed which became infected with a penicillin-sensitive *Staphylococcus aureus* and *Bacillus proteus*. Penicillin therapy was unable to sterilize this condition. A thoracotomy was performed on the eleventh day, and a broncho-pleural fistula was found as well as the fibrinohæmothorax. The patient was then evacuated to the mainland for further treatment. The amount of penicillin used was 200,000 units intrapleurally before thoracotomy, 300,000 units after thoracotomy (before the patient's transfer) and 800,000 units intramuscularly.

CASE V.—A rifle bullet traversed the left lung, causing a small sucking wound with a pneumothorax, but no apparent hæmothorax. The wound was excised and sutured eight hours later. Aspiration was not performed at first, as blood did not appear to be present on clinical examination. (X-ray facilities were not available to check the clinical findings.) However, 600 millilitres of blood were aspirated on the seventh day and a similar amount on the next day. In spite of further daily aspiration, a fibrinohæmothorax developed. Thoracotomy was performed on the eleventh day after receipt of the wound and a broncho-pleural fistula was found as well. Infection of the pleural contents did not occur. The patient was shortly afterwards evacuated to the mainland. The amount of penicillin used was 500,000 units into the chest and 570,000 units intramuscularly.

CASE VI.—This patient sustained a perforating rifle bullet wound from the right posterior triangle to the left axilla, traversing the apex of the right lung, the superior part of the mediastinum and the upper lobe of the left lung. As the bullet emerged, it shattered four rib shafts and the body of the scapula. The result was an apical pneumothorax on the right side, mediastinal emphysema and a massive hæmo-pneumothorax on the left side. The patient was suffering severely from loss of blood, and was distressed by dyspnœa and cyanosis from the increased tension in the left pleural cavity. In addition, numerous crepitations were heard in both lung fields, probably owng to the interstitial pulmonary emphysema resulting from the mediastinal emphysema.

The left pleural cavity was immediately aspirated, and in the next few hours over two litres of blood were removed. Aspiration and penicillin instillation were performed every second day; but a fibrinohæmothorax developed, which became infected with a penicillin-sensitive *Streptococcus hæmolyticus* and *Bacillus proteus*. The chest contents were unable to be sterilized, owing to the numerous loculi present. thoracotomy was performed on the eighteenth day and a little clot was again removed six days later. The chest condition was completely healed and the lung expanded four weeks later. The amount of penicillin used was 400,000 units intrapleurally before thoracotomy and 600,000 units after thoracotomy; 2,125,000 units were given intramuscularly.

CASE VII.—A submachine-gun bullet traversed the right lung, causing a hæmopneumothorax and an extravasation of blood into the lung. The parietal wound did not require excision. Aspiration was performed and penicillin was instilled within twenty-four hours of receipt of the wound. This was repeated every second day. On the fifth day a penicillin-sensitive *Staphylococcus aureus* was grown on culture from the fluid. (When the fluid was allowed to stand, the supernatant layer was seen to be pink in colour, a feature of early infection.) The fluid became a little turbid, but frank pus was never formed. Daily aspiration and instillation of penicillin were carried out. The pleural fluid became sterile three days later and the chest was free from fluid on the twentieth day. Twelve days later the lung fields were seen to be quite normal. The amount of penicillin used was 400,000 units into the chest and 450,000 units intramuscularly.

CASE VIII.—A rifle bullet traversed the neck, perforating the trachea and the left lung, and emerged from the left axilla, causing a large sucking wound with multiple rib injuries. A left hæmopneumothorax and mediastinal emphysema were present. The patient reached the surgeon eight hours later, when resuscitation measures were undertaken and tracheotomy was performed. He never rallied and died ten hours later. Autopsy confirmed the clinical findings; it revealed a little blood and air in the mediastinum as well as 1,500 millilitres of blood in the left pleural cavity. The left lung was collapsed and solid with extravasated blood, which was also present in the bronchi. The chest wall was infiltrated with much blood. The right lung was normal, and no blood was present in its bronchi.

CASE IX.—A mortar fragment penetrated the left pleural cavity and lodged in the upper lobe of the left lung. Enormous surgical emphysema of the head, neck, body and limbs developed, and when the patient was examined eight hours later he had a left-sided tension pneumothorax. The air was immediately released from the chest by a lumbar puncture needle, which was left *in situ*, and he was completely relieved of his respiratory distress, though numerous crepitations of pericardial type were still present behind the sternum. Eight hours later he suddenly collapsed and died. Autopsy revealed a left-sided pneumothorax under no tension. The lung, though collapsed, was crepitant with interstitial emphysema. A small piece of metal was found in the upper lobe of the lung, but no air could be expressed from the small wound of entry in the lung. A little air and blood were present in the mediastinum and had tracked through the hilum. There was no obvious rupture of the mediastinal pleura.

CASE X.—Multiple fragments penetrated the lateral aspect of the right side of the chest. One fragment smashed two ribs and caused a small sucking wound. The right pleural cavity was full of blood and air. Numerous small foreign bodies were present in the lower lobe of the right lung. One fragment had apparently traversed the anterior part of the mediastinum, because it was lodged in the lingula of the upper lobe of the left lung. There was no evidence of mediastinal emphysema.

The sucking wound was sutured in the front line, and the patient was examined by the surgeon twenty-four hours later, when the wounds were excised and successfully closed by primary suture. Aspiration was performed shortly after operation and thereafter every second day. The pleural cavity was free from fluid fourteen days later, and the lung was well expanded. The foreign bodies had caused no symptoms a month later. The amount of penicillin used was 50,000 units intrapleurally and 1,045,000 units intramuscularly.

CASE XI.—A submachine-gun bullet traversed the upper lobe of the right lung, fracturing two ribs *en route*. When examined two hours later, the patient had a tension pneumothorax and a mediastinal shift which caused him much respiratory distress and cyanosis. He complained of severe, constant pain over the right side of the abdomen, which was tender and rigid. There was no evidence of mediastinal emphysema. Immediate aspiration of air was performed, with dramatic relief of all symptoms, including the abdominal pain and rigidity. The parietal wound did not require surgical excision. A hæmothorax then developed, which was aspirated twice, so that the pleural cavity was free from fluid on the fifth day, and the lung was completely expanded on the eighteenth day. The amount of penicillin used was 100,000 units intrapleurally and 345,000 units intramuscularly.

Summary.

1. Thirty-two cases of gunshot wounds of the chest with intrapleural involvement are discussed. Two patients died within a few hours of being examined; 24 of the remaining 30 had blood in the pleural cavity.

2. The treatment of these patients was based on adequate resuscitation, early excision and suture of the wounds, early and repeated aspiration of hæmothoraces and early thoracotomy for fibrinohæmothorax and pleural infection.

3. Penicillin therapy, both intrapleural and intramuscular, was used as an adjuvant in all cases.

4. Complications included four cases of fibrinohæmothorax, in three of which infection supervened, the total of intrapleural infections thus being increased to five. Broncho-pleural fistula occurred in three cases and was responsible for two of the infections.

5. All wounds healed and the lungs expanded in six weeks, except in the three cases of broncho-pleural fistula.

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Reports of Cases.

A CASE OF PERIARTERITIS (POLYARTERITIS) NODOSA.

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PERIARTERITIS (POLYARTERITIS) NODOSA, a disease which affects all ages and both sexes, was until fairly recently thought to be inevitably fatal; but it was pointed out by Grant[1] in 1940 that this misconception arose merely because the condition was rarely diagnosed except in the autopsy room. He showed that of the patients whose condition was diagnosed during life quite a large proportion survived, and some of the patients have been followed long enough for it to be found that they died of quite unrelated conditions in later life. A study of the pathology of *periarteritis nodosa* provides grounds for this belief. The disease is a disseminated necrotizing arteritis of the smaller arteries, in which the essential lesion proceeds to natural healing if the patient survives. During the acute phase there is a tendency to hæmorrhage through the necrotic arterial wall, aneurysm formation of the arterial wall, which may rupture and bleed, and infarction of the tissue supplied by an affected artery, if this is completely occluded by thrombosis or by the pathological process itself, as it frequently is. There is also apparently some damage to the capillary walls, which explains the exudative phenomena. Now it is quite obvious that if these processes take place to any great extent in vital organs, such as the kidney or brain, death is most likely to result. If such organs escape or are affected only to a lesser degree, and if the brunt of the disease falls, say, in the skeletal muscles, the patient is likely to survive.

Spiegel,[4] and more recently McCall and Pennock,[2] have shown the relative frequency with which the various organs and systems are involved in subjects who come to autopsy. I know of no such analysis of non-fatal cases, but the figures would obviously be different.

With regard to the diagnosis, Spiegel[4] gave us something tangible when he pointed out that it is an acute febrile disease characterized by prostration, leucocytosis and tachycardia, to which is added evidence of circulatory disturbances in one or more organs or systems.

Obviously when only one organ is involved, or when a patient presents before more than one organ or system has become involved, diagnosis is extremely difficult if not impossible (see Spiegel's[4] and Grant's[1] series). But when the picture develops of scattered and unrelated circulatory disturbances, associated with the febrile condition described above, we have a characteristic disease which can be strongly suspected if not recognized clinically.

It was on these grounds that the diagnosis was suspected and later confirmed by biopsy in the case about to be described. At this point it may be wise to draw attention to the demonstration by Solomon, Kashich and Kiven[3] that negative biopsy findings do not eliminate the possibility of the disease.

Clinical Record.

L.B.P., a Royal Australian Air Force airman, aged eighteen years, was quite well until, after playing tennis on September 20, 1944, he noticed that his legs were stiff and painful. He went to the pictures that evening, and when he rose from his seat to go home found his legs so stiff and painful that he had difficulty in walking back to camp, one-quarter of a mile away. His legs were sore during the night and his sleep restless. All the next day his legs were "a bit stiff", but he slept fairly well that night. The following morning he woke at about 5.30 and vomited. The stiffness of his legs was wearing off, but he did not feel fit for work and he was allowed to spend the day lying about in his quarters. He ate nothing

during the day, vomited twice more in the evening and had another restless night. The next morning (the fourth day of his illness) he was admitted to the unit sick quarters, where he also complained of pain in the back (upper lumbar region). The notes record that on examination on his admission to hospital his temperature was 97° F. and his pulse rate 88 per minute, and that his respirations numbered 20 per minute. He was pale, his tongue was coated with a brown fur and his breath was offensive. No abnormality was detected in the heart and lungs. Slight epigastric tenderness was present, and pronounced tenderness was elicited in both renal areas at the back. His urine was dark and scanty; it contained a heavy cloud of albumin, and on microscopic examination numerous erythrocytes were found in it.

The next day a painful swelling of his right forearm from the elbow downwards developed. This persisted until the following day, when the amounts of albumin and blood in his urine had increased, and he was transferred to a Royal Australian Air Force base hospital during the afternoon of September 25, with the diagnosis of acute nephritis. On his arrival, further questioning revealed that he had had a severe sore throat three weeks prior to the onset of his illness. He had had no other serious illnesses in his life.

On examination of the patient his temperature was 102·2° F. and his pulse rate 106 per minute, and his respirations numbered 30 per minute. His throat was not congested, and the tonsillar fossæ were clean (his tonsils having been enucleated about twelve years previously). The heart and lungs were normal; but his blood pressure was 164 millimetres of mercury, systolic, and 110 millimetres of mercury, diastolic. The right wrist and metacarpo-phalangeal joints were red, hot, swollen and tender, and stiffness and tenderness of the right elbow and shoulder were present. There was no oliguria, and on macroscopic examination the urine was found to contain no macroscopic blood, but a "heavy cloud" of albumin. Microscopically it was found to contain a heavy deposit of amorphous debris, about six to eight erythrocytes and one or two leucocytes per high-power field. No casts were seen. A provisional diagnosis was made of acute rheumatic fever with concomitant acute nephritis. Treatment with full doses of sodium salicylate was begun. During the next twenty-four hours both his ankles, both knees and the right shoulder became swollen and painful, and the swellings in his right forearm decreased.

By the fourth day in hospital (September 28) his temperature had fallen to normal and the pain had disappeared from his joints. This was interpreted as a response to salicylates, strengthening the diagnosis of acute rheumatic fever. The amount of albumin in the urine was reduced to a light cloud only. The blood picture was as follows: the hæmoglobin value was 13·7 grammes *per centum* and the leucocytes numbered 19,000 per cubic millimetre, 91% being neutrophile cells. The erythrocyte sedimentation rate was 32 millimetres per hour (Westergren). Later in the day, however, the patient began vomiting large amounts of fluid in which specks of blood were seen, and in one specimen a clot of blood (about half an ounce) was found. On the following morning (September 29) the patient passed a black stool, and the continued vomiting made it necessary to commence intravenous therapy. A total of three litres of wet serum and one litre of glucose in saline solution was given over approximately twenty-four hours. By the end of this time the patient had ceased vomiting, was drinking fluids, and showed clinical signs of restored body fluid balance, and his blood protein, chloride and hæmatocrit levels were back to normal range. The prostration at the height of this episode had been extreme, and although the apprehension concerning his immediate prognosis was allayed by his response to the venoclysis, all his muscles were rapidly wasting. He lay motionless in his bed throughout the acute phase of his illness, was lethargic and seemed peculiarly indifferent to his condition.

The bleeding and clotting times were within the normal range and the prothrombin level was 60% of normal. The blood picture on September 30 was as follows: the erythrocytes numbered 4,000,000 per cubic millimetre, the hæmoglobin value was 12·5 grammes *per centum*, and the leucocytes numbered 21,000 per cubic millimetre, 73% being neutrophile cells, 21% lymphocytes, 1% eosinophile cells and 5% monocytes. On the same day microscopic examination of the urine revealed 50 to 60 erythrocytes and four or five leucocytes per high-power field, but no casts.

The next phase of his illness began on the evening of September 31, with a rise of temperature to 102° F. The temperature oscillated between 100° F. and 103° F. during the next three days and then fell to normal again. During this time his lethargy increased, his tongue became dirty and his abdomen a little distended, and he was nauseated but did not vomit. On the fourth day (October 3) he passed three fairly large, dark-red fluid stools. They contained almost no fæcal matter and consisted almost entirely of pure blood. He passed two more similar stools during the night.

On October 3 the leucocyte count rose to 58,000 per cubic millimetre, 94% being neutrophile cells, 0·5% eosinophile cells, 4% lymphocytes, and 1·5% monocytes. As the number of leucocytes was so high the count was repeated the next morning, when 51,000 leucocytes per cubic millimetre were counted.

At this time moderate numbers of hyaline and granular casts were seen in the urine for the first time. Blood taken for cultural examination during this febrile phase yielded no growth of organisms. The Widal and Weil-Felix tests produced no reactions.

The next phase began on October 6, when his temperature rose to 103° F. and he complained of severe pain in the anterior aspects of both arms. Examination revealed only severe tenderness of the biceps and brachialis muscles on both sides.

During the following few days the fever persisted and the volar aspects of both arms became swollen, tense, hard and tender. The patient was quite incapable of flexing his elbows at all—not so much, I think, because of the pain, but because the swollen muscles "just would not work". The leucocytes numbered 25,000 per cubic millimetre, 92% being neutrophile cells.

By October 11 the fever and the swellings had subsided. There was no residual bruising and no evidence of extravasated blood. From October 11 to 22 an irregular pyrexia was present, with evening rises of temperature to about 100° F. and occasionally 101° F. Throughout this time the general condition of the patient gave no cause for concern, but he remained emaciated. The predominantly interesting clinical feature, however, was the appearance in the urine of masses of pus cells in the centrifuged deposits. (There were a few associated erythrocytes and hyaline and granular casts.) Microscopic examination of the urine was made every day and revealed the same picture until November 4; on that day the findings suddenly fell to four to six erythrocytes and one or two leucocytes per high-power field and a few hyaline casts (and this was representative of the findings until his transfer from hospital on January 2, 1945—*vide infra*). The interesting feature of this period of pyuria was that cultural examinations produced at times no growth or on some occasions only innocuous organisms and there was no clinical evidence of urinary infection. The pus cells were presumed to be coming from the lesions in the kidney during the stage of repair.

A biopsy of the right *biceps brachialis* taken on October 25, 1944, showed the histological picture of healing *periarteritis nodosa*.

On November 3, 1944, the report was obtained that blood from the patient, submitted on about October 12, did not agglutinate four strains of *Leptospira icterohæmorrhagica*.

Other investigations carried out were as follows. On October 6 X-ray examination of the heart and lungs revealed no abnormality. On November 21 an electrocardiogram showed no significant abnormality. The leucocyte count gradually fell and was within normal limits by October 12. There was no eosinophilia at any stage. No

abnormalities were found at any stage in the eye grounds. The blood pressure readings fell to about 130 millimetres of mercury, systolic, and 90 millimetres of mercury, diastolic, by about the end of October, and have not shown any significant elevation since then.

By early January, 1945, the patient was well and was putting on a lot of weight. He was up and walking about; his urine contained no albumin at times and at others a cloud of albumin, and in the centrifuged deposit were found two or three erythrocytes and an occasional leucocyte per high-power field and no casts. On January 2, 1945, he was transferred to a Royal Australian Air Force medical rehabilitation unit, where he remained until February 14. While there he had a slight swelling of his left wrist, which was a little painful and lasted for one day only, and on another occasion pain in both loins, lasting for one day. Apart from this he was well during his stay. His urine was examined twice a week for albumin, but none was present on any occasion.

On his return to hospital for review on February 14 he had no symptoms, general physical examination revealed no abnormality, the fundi were normal, the blood pressure was 140 millimetres of mercury, systolic, and 80 millimetres of mercury, diastolic, and the urine contained no albumin. Microscopic examination of the centrifuged deposit of the urine revealed no abnormality.

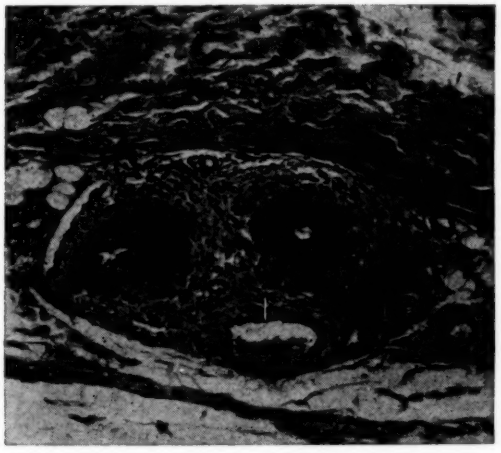

FIGURE I.

Skin and subcutis of the arm. Sections of biceps muscle and of the adjacent skin were stained with Ehrlich's hæmatoxylin and eosin and with Weigert's iron hæmatoxylin and Van Gieson's collagen stain, combined with Weigert's elastic stain. Most of the vessels appear histologically normal. A few show an excess of laminated collagen tissue in the adventitia, the significance of which is doubtful. One group of vessels in the subcutis shows distinct pathological changes. The group consists of two terminal arteries and a small venule. These vessels are set in a background of granulation tissue composed of leucocytes, lymphocytes, plasma cells, fibroblasts and young collagen fibres. In one artery the media, for three-quarters of its circumference, is replaced by an eosinophilic exudate containing leucocytes and indistinct red corpuscles. The intimal endothelium is swollen and several nuclei are pyknotic. In the other artery the media is reduced to a single layer of stretched muscle fibres, internal to which is a broad zone of eosinophilic colloidal exudate containing a few nuclear remnants and bounded internally by the swollen endothelial cells of the intima. The venule appears normal. These histological changes are typical of active *periarteritis nodosa*.

A urea concentration test was performed on February 16, but the value of the test was upset by the diuretic effect of the urea. It was therefore repeated on February 23, when the maximum concentration of urea in the urine was three grammes *per centum*. Cytological examination of the urine on this date revealed 10 to 15 erythrocytes and four or five leucocytes per high-power field, and scanty epithelial cells. The fasting blood urea level was 28 milligrammes per 100 cubic centimetres of blood.

On February 27 the patient complained of pain in the right loin, and on examination he was found to be tender to palpation over the right kidney area. Examination of the urine revealed a light cloud of albumin, but unfortunately no microscopic examination was made on this occasion. This pain disappeared within three days, but the albuminuria has persisted since; it varied in intensity from a light cloud to a trace, and on some days none.

Microscopic examination of the centrifuged deposit of the urine on March 13, 1945, revealed one or two red cells per high-power field, some crystalline fragments and epithelial cells and mucus; no casts were seen. This is the position at the time of writing. The patient feels well and is putting on weight, and his blood pressure is 140 millimetres of mercury, systolic, and 80 millimetres of mercury, diastolic. He is up and about, but is still being retained under observation in hospital.

Discussion.

It is realized that it is too early to state that this patient has recovered from *periarteritis nodosa*; but as circumstances will not permit me to observe him clinically beyond this stage, the history is presented for what it is worth. He has, however, survived the acute diffuse stage of his disease, and after six months (at the time of writing) is relatively well. It is hoped that the occasional episodes of muscle pain and slight hæmaturia which have occurred over the last month or so represent the dying flickers of an almost burnt-out disease process. This may be over-optimistic.

The diagnosis in the case was a problem from the beginning, when rheumatic fever and acute nephritis were thought to be coexisting. The occurrences of gastro-intestinal hæmorrhage complicated the picture considerably, and *periarteritis nodosa* was first considered as a possibility at this stage, but with no real confidence, and every endeavour was made to exclude all other possibilities—generalized capillary disease secondary to streptococcal throat, septicæmia, the hæmorrhagic diseases, Weil's disease *et cetera*. The occurrence of the exudates into the muscles, with the unusually high leucocytosis, made *periarteritis nodosa* much more likely—especially as the literature had been more closely consulted by this time.

The ætiology of the disease is unknown, but as allergy and infection both have their advocates, it is worth drawing attention (*a*) to the fact that the patient under discussion suffered from an acute sore throat (probably streptococcal) three weeks prior to the onset of his illness, and (*b*) to the information obtained from the patient's mother that when the patient was a child, if he ate walnuts or snapper (but not other fish), his "eyes would swell enormously and would not go down unless the doctor came and gave him an injection". There was no history of any other allergic manifestation.

Summary.

1. A case of *periarteritis nodosa* has been described. The patient was a young male adult, who presented the syndrome of an acute febrile illness, with prostration, high leucocytosis and tachycardia associated with renal manifestations indistinguishable from acute nephritis, acute painful swellings of the muscles and joints, hæmatemesis and melena.

2. The diagnosis was confirmed by biopsy of the *musculus biceps brachialis*.

3. The acute phase of the disease has subsided and the patient is still reasonably well after approximately six months.

4. The diagnosis and ætiology are briefly discussed.

Acknowledgements.

I wish to thank the Director-General of Medical Services, Royal Australian Air Force, for permission to publish this case; Dr. F. B. Byrom, Director of Post-Graduate Pathology, University of Sydney, for having prepared and reported on the specially stained sections from which was taken the photomicrograph published; and to Mr. Woodward Smith for the preparation of the photomicro-

graph. I desire also to acknowledge the work performed by the late Major L. Utz in preparing and reporting on the biopsy specimens.

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LOCAL AMPUTATION OF GANGRENOUS TOES IN THE PRESENCE OF GLYCOSURIA AND SENILITY.

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The following cases are believed by the writer to be evidence in favour of the abandonment of the generally accepted idea that in cases of diabetic gangrene of the toes a high amputation of the leg is necessary.

Case I.

A.H., aged sixty-nine years, a bailiff, was a large man, weighing 220 pounds. He was first examined on November 20, 1944, suffering from inflammation of the sole of the foot close to the base of the second right toe. The condition was associated with suppuration underneath a callus which the patient had cut a day or two previously. The toe at the time was mildly congested. The patient was admitted to hospital, and a *régime* of sulphanilamide therapy was commenced. Subsequently I learned that heavy glycosuria was present, and the patient then gave a history which suggested to me that for years he had known himself to be a sufferer from diabetes. His statement was accepted, and 20 units of insulin were ordered to be given before his lunch and 20 units of protamine zinc insulin to be given at 4 p.m.

On November 23 almost the whole of the toe was gangrenous. The next day his temperature was 101·2° F. Penicillin treatment was commenced, 15,000 units being given every three hours. As severe glycosuria was still present on the following day, the foregoing doses of insulin were doubled. On November 26 his temperature was much reduced, and sulphanilamide treatment was stopped. Three days later the application of wet dressings was discontinued, and the foot was exposed to radiant heat (whilst elevated under a cradle) for half an hour in every two hours. The patient was instructed frequently to undertake inverted "bicycle" exercises.

By December 1, the course of penicillin treatment (800,000 units) was completed. Six days later much cyanosis of the dorsum of the foot was present in the form of a triangle, the centre of the base being at the base of the gangrenous toe and the apex high up the inner side of the instep. On December 9 the apex of this triangle was incised and much pus was obtained from the superficial layers of the subcutaneous tissue. The next day the patient was instructed to spend twenty minutes twice a day with his leg up to the thigh in a "Both" cabinet respirator, the exit valve of which was partially closed so as to give a "swing" on the manometer of from 0 to 24 (approximately). Sulphathiazole therapy was commenced, and two days later the inflammation was declining; the line of demarcation between the base of the toe and the remainder of the foot was becoming well defined. On December 18 penicillin treatment was recommenced.

Four days later, three capsules of "Nembutal" were given, and two hours later a femoral periarterial sympathectomy was performed, in which three and a half inches of the artery were denuded; 5,000 units of penicillin were placed in the wound. After four or five hours' initial cooling of the extremity it became much warmer to the touch than its opposite number, and remained so until the patient's discharge from hospital. A course of penicillin treatment was commenced prior to the operation, as it was thought that the infliction of any surgical trauma along the lines of the lymphatics from a suppurating area would be courting trouble; the thigh healed by first intention.

On December 23, suppurating and sloughing areas at the base of the toe were irrigated with a little penicillin solution. The next day the leg was hung over the side of the bed for a few moments at frequent intervals. On the following day, when 800,000 units of penicillin had been given, sulphathiazole and penicillin treatment was discontinued; the discharge from the base of the toe was diminishing. On December 31 there was no further discharge from the instep.

On January 2, 1945, the healed sinus of the instep reopened, and much discharge was present. The patient began walking on crutches, without weight-bearing on the foot. Four days later the discharge was more profuse from both the base of the toe and the sinus on the dorsum of the foot. On January 11 X-ray examination revealed pronounced osteoporosis of the phalanges of the toe and of the head of the corresponding metatarsal. Five days later a further course of penicillin treatment (1,000,000 units) was commenced. The dorsum of the foot later became inflamed and œdematous, and a further course of sulphathiazole was begun. Another X-ray examination revealed advancement of the previous condition, but no spread. On January 26 the condition of the foot was improving, and next day sulphathiazole treatment was stopped.

On February 8 a pre-operative course of penicillin therapy was commenced, and next day (six weeks after sympathectomy), under anæsthesia with "Avertin" and ether given by the "open" method, a wedge was amputated so as to include the toe and the head of the metatarsal. A long, fibrous slough leading up to the incision on the dorsum of the foot was pulled out of the depths of the wound. There was disturbingly little hæmorrhage, all the tissues being firmly œdematous. (No ties or sutures were used.) Sulphanilamide powder was placed in the wound. One week later penicillin treatment was discontinued, and on March 9 the patient was discharged from hospital. On March 15, five weeks after amputation and approximately four months from the onset of the disability, the wound was healed. The patient remains in good health.

Comment.

The various methods of physical therapy utilized in this case (elevation, intermittent radiant heat under a cradle, "bicycle" exercises, the use of the "iron lung" and posturing) were kept up for the whole of the time during which the patient was in hospital, except for one or two brief breaks.

The amounts of insulin given in this case were sufficient to keep the patient's urine practically sugar free for most of the time he was under treatment. Subsequently a blood sugar curve showed no departure from the normal.

Case II.

T.G., aged seventy-two years, a locksmith and gunsmith, weighed 217 pounds. He was known since July, 1943, to be a diabetic. When examined on March 20, 1945, he had superficial gangrene of the terminal phalanx of the left fourth toe, following an abrasion from a nail in his shoe some days before. The foot about the toe was inflamed. His temperature was 99·8° F.

Treatment with sulphathiazole in heavy dosage was commenced immediately, as also was the administration of penicillin, 15,000 units every three hours. The foot was elevated under a cradle and intermittent heat was applied. The insulin *régime* instituted was as follows: 6.30 a.m., insulin, 5 units; 4 p.m., protamine zinc insulin, 16 units.

On March 21 the toe looked drier, and a bleb was removed; the patient's temperature was still elevated, and

the next day it was 100·4° F.; he was drowsy. The dosage of protamine zinc insulin was reduced to 12 units. Two days later his temperature was normal. His toe was thereafter painted with *Spiritus Methylatus* and "bicycle" exercises were instituted; penicillin treatment was stopped. One week later periarterial sympathectomy was performed under local anæsthesia; four inches of the artery were denuded, and 5,000 units of penicillin were put in the wound. The leg was much warmer in four hours. Six days later some dead skin was removed from the toe, and six days later again the patient began treatment with *Thyreoidum Siccum*, one grain three times a day.

On April 16, under anæsthesia with "Avertin" and with ether given by the "open" method, the toe was amputated, treatment with penicillin having been commenced nine hours before. One suture was used, and sulphanilamide powder was put in the wound. On April 19 penicillin treatment was stopped, and on April 22 the wound was healing by first intention. On April 27 the patient went back to work, five and a half weeks after his admission to hospital. The foot has given no trouble since.

Reviews.

NEUROPATHOLOGY.

THERE are some authors whose later editions are so enlarged to keep pace with the times that friends of the first edition scarcely recognize their old authority. We must congratulate Professor Weil because the second edition of his well-known textbook on neuropathology[1] quite escapes this censure in that with 27 new illustrations and a like number of pages, he has managed to include the more important new conceptions and accretions of knowledge added to neuropathology since the first edition some twelve years ago.

The enormous increase in the operations of neurosurgeons and the increasing demands by physicians as well as neurologists for reports on the nervous systems of their patients have resulted in the examination of the brain in all possible cases, in the preparation of a slide or two, and when a definite neurological history is available, in the carrying out of proper system examinations. One result of this system is that conditions previously rated rare have now become familiar and greater prominence is being given to this subject in scientific journals; the neuropathologists, too, can give more confident reports.

However, while we may pride ourselves on our aid given to the neurosurgeon with biopsies, we must still acknowledge that we have advanced but little in relation to mental diseases. Professor Weil includes a fair account of changes in the brain in connexion with the convulsive therapies and is careful to point out that the microscope alone cannot be the complete judge. Clinical phenomena, vitamin properties, chemical effects and changes in the tissues must all be considered, as well as the knowledge derived from carefully planned and widespread experiments on animals. Finally the provision of cool chambers eliminates much of the dreaded post-mortem change so fatal to useful reports in this field.

In spite of new work on the Schwann cell we still remain somewhat confused about the real constituents of a nerve sheath tumour, and while much care is bestowed on the encephalomyelitides, their classification has not been greatly simplified. The author has studied minutely cases from epidemics in both man and animals, including the St. Louis types and those from Japan and Australia, and due acknowledgement is made to Hurst and his school.

Weil makes a point of stressing the value of the newer silver impregnations in revealing further structure in well-known lesions. By this the argentophile network in the centre of gummata distinguishes them from tuberculomata, and in other conditions provides an additional conception of their intimate structure; at times also it provides a means of classification. Tabes still defies our best intentions, but these methods bring us nearer our goal in elucidating certain reactions, newly recognized tissues and debris in the old posterior root regions. Giekie Cobb and his school are appealed to when the physiology of the capillaries and their permeabilities are discussed, especially in relation to endothelial damage in infections, intoxications and similar states. Naturally the author has his favourite subjects which are dealt with quite fully, as, for instance, inflammations and infections including the blastomycetes. The chapter dealing with the intoxications gives a good summary of the brain reactions after the exhibition of metrazol, insulin and the barbiturates. Those who may be interested in cerebellar phenomena will be disappointed, since this interesting chapter in neuropathology is but scantily alluded to under the degenerative states. However, as a whole the presentation of the degenerations provides a good test of the real value of this book to neuropathologists.

The chapter on tumours, which has always been a feature, is doubly so in this edition because of its reference to the extraordinarily interesting light thrown on tumours of the nervous system by their artificial induction in animals by the use of carcinogenic material. By these agents normal neuroglia may be seen turning into spongioblasts, several types of tumours eventuate from the dura, and a real tumour is obtained from the posterior or *pars nervosa* of the pituitary—a phenomenon we are awaiting in nature. The author himself has covered much ground in this field.

Similarly peripheral nerve sheath tumours may be seen to be combinations of neoplastic Schwann cells and neoplastic fibroblasts. The neoplastic transformation of whole organs—medulla, pons, lobe of brain which often intrigued us—is made understandable by reference to liver carcinoma induced by feeding butter yellow. A short account of congenital malformation covers much ground. The author's method for staining myelin fibres for paraffin, celloidin and frozen sections has proved excellent in our hands and has been copied in many modern manuals.

This *résumé* of the book amply reveals its value to the practising neuropathologist and student, and again stress must be laid on the illustrations and tables.

[1] "Textbook of Neuropathology", by Arthur Weil, M.D., Associate Professor of Neuropathology, Northwestern University Medical School; Second Edition; 1945. New York: Grune and Stratton; Sydney: Angus and Robertson, Limited. 9" × 6", pp. 372, with 289 illustrations. Price: $5.00.

PROGRESS IN OBSTETRICS AND GYNÆCOLOGY.

THE sixth edition of "Recent Advances in Obstetrics and Gynæcology" just published maintains the high standard of previous editions, and we strongly recommend it to specialists and general practitioners interested in this branch of medicine.[1] It should prove of special value to those younger members of our profession who have spent several years in the services, and have thus been almost completely out of touch with this work. The editors have discarded certain sections which have no new knowledge to be recorded and can be referred to in earlier editions, and have replaced them by several new chapters of first importance. Other sections have been partly rewritten and brought up to date by the addition of new material.

The chapter on nutrition in pregnancy and fœtal development replaces, or rather amplifies, the earlier chapter on ante-natal care. Fuller discussions are given on the optimum diet of pregnancy; food values; vitamins; effects on the mother, and particularly on the fœtus, of malnutrition during pregnancy; faulty cooking; storage of foods *et cetera*. Tables are given showing the very wide variations of values in minerals and vitamins for the same foodstuffs. From these it is clear that in future the agriculturist will have to play his part in producing food of the best quality to help in the prophylaxis and treatment of disease, and so to improve the national standard of physique and health.

The defect in the last edition that no reference was made to vitamin *K* and hæmorrhagic disease of the newborn has been rectified in this edition. One of the new chapters deals with hæmorrhagic diseases of the newborn and their probable causes and treatment. It is agreed that vitamin *K* should always be given as a prophylactic to the mother who is having a premature baby, and in cases of prolonged or difficult labour, to raise the prothrombin level of the fœtus. The authors also urge that in the actual treatment of hæmorrhagic disease it is wise in all cases to use the older method of transfusion of fresh blood as well as to give vitamin *K*,

[1] "Recent Advances in Obstetrics and Gynæcology", by Aleck W. Bourne, M.A., M.B., B.Ch. (Camb.), F.R.C.S. (England), F.R.C.O.G., and Leslie H. Williams, M.D., M.S. (London), F.R.C.S. (England), F.R.C.O.G.; Sixth Edition; 1945. London: J. and A. Churchill Limited. 8" × 5¼", pp. 367, with 77 illustrations. Price: 18s.

since some cases may be due to lack of thrombokinase or fibrinogen.

Discussions on the use and misuse of the many anæsthetics and analgesics in labour and Cæsarean section have been amplified. In particular the value of the new analgesic pethidine and the advantages and disadvantages (and dangers) of caudal anæsthesia are dealt with. Continuous caudal technique is fully described, and while admitting its limitations and certain objections to its use, the authors express the hope that the method will be given an extensive trial in those British obstetric hospitals which have the requisite whole-time obstetric and anæsthetic staff, as well as highly trained nursing personnel. They agree with Professor Francis Browne and other authorities that it can never come into general use in domiciliary or small hospital practice.

The increasing urgency of the falling birth rate in the Western world and the importance of making every effort to prevent the birth of dead babies and to save those born alive are stressed in the new chapter on stillbirth and neo-natal death. Over 50% of neo-natal deaths are due to prematurity, and it is important that all immature babies should have proper care by a pædiatrician and by nurses who have had special training. The Ministry of Health, on the advice of its technical committee, recommends notification of all babies under five and a half pounds, and the formation of special teams and facilities to ensure their prompt and efficient care. Among suggestions for lowering the incidence of stillbirth and neo-natal death is a plea to avoid induction of premature labour whenever possible; the authors consider this is done unnecessarily often.

In the gynæcological section a useful summary has been added covering the procedures which form a complete investigation in a case of sterility.

Bates and Judovich's intrathecal injection of ammonium sulphate instead of alcohol is becoming increasingly popular for the relief of pain in pelvic cancer, but is still on trial. Sympathetic nerve block for *phlegmasia alba dolens* constitutes a really valuable advance in therapy, and dicoumarin, a new synthetic anti-thrombotic substance, which inhibits the formation of prothrombin by the liver, is also on trial. It can be given orally or intravenously, either prophlactically or for treatment in already established cases of venous thrombosis. Its action is slower but more prolonged than that of heparin given intravenously, but they may be used together. It is necessary to remember a possible danger in this form of treatment which may keep the prothrombin time elevated from three to seven days after the drug is discontinued.

There are only some minor additions to the section on sex hormones, as in spite of an extraordinary amount of biochemical experimental work on animals, there has been little real advance on the problems of sex endocrinology. Wilfred Shaw's contribution has some additional material, mainly an interesting review of modern theories on the origin of ovarian tumours. Though these tumours still provide an "insoluble problem", considerable advances have been made, and the histogenesis of different tumours has been more clearly established, while experimental embryology has in recent years brought to light influences which are of great importance in the development of ovarian tumours.

The excellent section on radiology in obstetrics has heeded a little alteration, and is still considered to be the best short account available.

There is a striking change in the introductory sentence to the section on cancer of the uterus. "We feel that medical opinion the world over has now crystallized and that it should definitely be stated that the surgical treatment of cancer of the cervix has been superseded by radium treatment combined, when advisable, with deep X-ray therapy." The only reservation made is that there is still a place for surgery in Stage I or Stage II cases in which there is an initial failure to respond to radium. Judgement of this failure to respond is said to be aided by a series of biopsies before and during treatment, as described by Donaldson (1943). Such cases must be few, but the possibility is worth remembering.

It is particularly satisfactory to notice the emphasis which is given to the necessity for team work in improving results.

An additional chapter on X-ray therapy in gynæcology has been contributed by W. M. Levitt. It should be useful for the gynæcological student to whom it is addressed, but it seems to suffer from over-simplification and contraction. The author's personal choice of preliminary X-ray therapy before radium in the more advanced cases of cancer of the cervix is, as he points out, unusual, and indeed the whole question of attempting radical treatment in Stage IV cases should not be lightly decided.

We congratulate the editors, who have attained their objects and have presented a volume of about the same size as its predecessor which contains most of the important new knowledge on obstetrics and gynæcology.

THE PSYCHOLOGY OF WOMEN.

HELENE DEUTCH'S "The Psychology of Women" differs from most books on psychoanalysis in that it is original in its conceptions, and eschews the use of neologisms.[1] Deutch, for many years a pupil of and co-worker with Freud, unlike most women psychoanalysts, writes scientifically and impartially about her own sex. She is ambitious and seeks to analyse the conflicts and emotions of women from infancy to old age. The completed work will be in two volumes, of which this is Volume I. The preoccupation of women with sex is, as can be expected, the predominant theme of the book: the development of sex from the phantasies of infancy and childhood to its full maturity in the normal woman, or to its maldevelopment into such aberrant forms as masturbation, masochism or homosexuality. The influence of parents, home, companions, environment and experiences in moulding a woman's thoughts and ideals is traced in detail. Much of this has been written before, but never so comprehensively. In fact the subject matter is so great that the book must be read and reread.

The author's clarity of thought and simplicity of language betray the influence of Freud; the very long and elaborate case histories, the influence of her present American environment.

Many of her observations are worth repeating. How true is her summary of promiscuous young girls during the war years: ". . . they play and act as young people of their age have always done, only the reality of the war has given their behaviour a more dangerous form. . . . As a result of their weakened self-control and psychologic instability immature girls are exposed to serious personal dangers, and as a mass phenomenon create an almost insoluble social problem" (page 53).

The book is beautifully bound, its bibliography is extensive, all in keeping with one of the best books on psychoanalysis that has appeared for many a day.

NURSING IN ACUTE INFECTIOUS DISEASES.

IN the 1945 edition, Dr. Scholes's excellent book on nursing in acute infectious diseases has been brought up to date, in spite of the difficulties associated with war conditions.[2] Information has been added on the important additions made to medical knowledge in the six years since the first edition appeared. The section on chemotherapy has been rewritten, and includes the newer members of the sulphonamide group of drugs. An excellent feature is the description of the undesirable symptoms which may come to the notice of the nurse after administration, and the question of individual susceptibility. Penicillin is described, and some of its uses are noted; no doubt in a later edition, when the administration has been subjected to more general experience, it will be possible to give more detailed instructions to nurses on the details of the technique of penicillin injection. The book is admirably printed and somewhat less bulky than the previous edition. A very good feature which could be copied with advantage by other publications is the use of heavy type for the principal names and important phrases; this is an aid to study and memory. General practitioners and nurses should have this book for reference, and the author is to be congratulated upon a very sound and comprehensive work.

[1] "The Psychology of Women: A Psychoanalytic Interpretation", by Helene Deutsch, M.D., with a foreword by Stanley Cobb, M.D., Volume I; 1944. New York: Grune and Stratton. Sydney: Angus and Robertson Limited. 8¼" × 5¾", pp. 411. Price: 33s. 6d.

[2] "Nursing in Acute Infectious Disease", by Frank V. G. Scholes, C.M.G., M.D., B.S. (Melbourne), D.P.H. (Cambridge), F.R.A.C.P.; Second Edition; 1945. Sydney: Australasian Medical Publishing Company, Limited. 8¼" × 5¼", pp. 338. Price: 16s.

The Medical Journal of Australia

SATURDAY, MARCH 2, 1946.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

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DISCUSSIONS ON HEALTH LEGISLATION IN THE UNITED STATES OF AMERICA.

WHILE Australian medical practitioners, interested in the provision of an adequate health service for the community, will easily recall the many discussions held during recent years by the Federal Council of the British Medical Association, the State Branches of that body, the National Health and Medical Research Council, the Parliamentary Joint Committee on Social Security, representatives of the Commonwealth Government, and other groups, they may not all realize that the subject has been engaging the attention of the medical profession and of the legislature in many other countries. Indeed it may be said that discussions on national health are a logical outcome of the war but recently ended. Reasons to support such a statement could be advanced without difficulty by any thoughtful member of the community. If Australians will take the trouble to look into the happenings in other countries, they may see their own problems more clearly, and they should, if they lack such a conviction, be convinced of the urgent need to find a solution. In the past attention has been drawn in these pages to plans and discussions that have been before the medical profession in Great Britain; some recent happenings in the United States should be brought to the notice of readers. The information concerning them will be found in *The Journal of the American Medical Association* of November 24, December 1 and December 8, 1945.

On November 19, 1945, President Truman submitted to the United States Congress a special message on the health of the nation. It was in effect a programme on national health. He stated that in the past the benefits of modern medical science had not been enjoyed by citizens with any degree of equality, nor were they today. They would not be equally enjoyed in the future unless the Government was bold enough to do something about it. The President held that the health of the nation was a national concern, that financial barriers in the way of attainment of health should be removed and that the health of all its citizens deserved the help of all the nation. He named five basic problems which had to be vigorously attacked if the health objectives of their economic bill of rights were to be reached. The first had to do with the number and distribution of doctors and hospitals. The United States had been fortunate in regard to medical practitioners ("physicians" in American parlance); in proportion to population it had more than any large country in the world, and they were well trained; distribution, however, was grossly uneven and unsatisfactory. The President's second basic problem was the need for the development of public health services and maternal and child care. He pointed out that services for expectant mothers and for infants, for the care of crippled or physically handicapped children, and for inoculation against communicable diseases were not available to a large part of the population, and that this also applied to many personal services, such as the diagnosis and treatment of such widespread infections as tuberculosis and venereal disease. The third problem concerned medical research and professional education. In this regard he had something to say about mental disease, for mental patients occupied more than half the hospital beds in the United States. The fourth problem had to do with the high cost of individual medical care. The chief reason why people did not receive the care that they needed was that they could not afford to pay for it on an individual basis at the time they needed it. This was true not only for needy persons, but also for a large proportion of those who were normally self-supporting. Only about 4% of the national income was spent on health services and the nation had to afford to spend more. The fifth problem was concerned with loss of earnings when sickness occurred. Sickness not only brought doctor's bills; it also cut off income. To solve these problems the President made certain suggestions, embodying a health programme, each part of which contributed to all the others. He advocated the construction of hospitals and related facilities, the expansion of public health, maternal and health services, the strengthening of medical education and research and the repayment of medical costs. Medical costs, he thought, should be paid for by expansion of the existing compulsory social insurance system. Needy persons and other groups should be covered by appropriate premiums paid for them by public agencies. The President insisted that under such an arrangement people would be free to choose their own medical attendants and hospitals, and medical practitioners should remain free to accept or reject patients. He also declared that what he was recommending was not "socialized medicine". The President's views on the administration of a nation-wide system are interesting. Since medical services are personal, the administration of a nation-wide system must be "highly decentralized". "The local administration unit must be the keystone of the system so as to provide for local services and adaptation to local needs and conditions. Locally as well as nationally, policy and administration should be guided by advisory committees in which the public and the medical professions are represented."

On the same day on which President Truman presented his health programme, Senator Wagner for himself and Senator Murray introduced to Congress a bill proposing to enact a *National Health Act of* 1945. A companion bill was introduced on the same day into the House of Representatives by Representative Dingell. An analysis of the bill has been prepared by the Bureau of Legal Medicine and Legislation of the American Medical Association. The bill contains two titles. The first is "Grants to States for Health Services". In this are three parts, dealing respectively with (*a*) grants to States for public health services, (*b*) maternal and child health services, and (*c*) grants to States for the medical care of needy persons. The second title is "Prepaid Personal Health Service Benefits". It is proposed to make available "personal health service benefits" to certain groups of persons and their dependants. The service will be paid for out of a "Personal Health Services Account" which will be created at the Treasury Department. "Personal service health benefits" include general medical benefits, special medical benefits, general dental benefits, special dental benefits, home nursing benefits, laboratory benefits and hospitalization benefits. A National Advisory Medical Policy Council will be created and will consist of the Surgeon-General as chairman and sixteen members appointed by him and approved by the Federal Security Administrator. The appointed members will be selected from panels of names submitted by professional and other interested organizations; representatives of the public will be included. Panels of patients will be drawn up for practitioners (general practitioners, specialists and consultants) who agree to work under the scheme. General practitioners will be paid: (*a*) on a fee-for-service basis, (*b*) on a *per capita* basis, (*c*) on a salary basis, or (*d*) on a combination or modification of these bases. The method of payment will be determined in each area as the majority of practitioners may elect. Specialists and consultants will be paid, as the Surgeon-General and the consultants may agree, on the basis of salary, per session, fee-for-service, *per capita* or other basis. In each local area the provision of general medical benefits is to be a collective responsibility of all general practitioners who have agreed to furnish the benefits.

The American Medical Association in its journal describes this as "government by minority with a vengeance". It points out that it was not consulted, and that it has a membership of more than 125,000 American doctors. It describes the proposals as involving both socialized medicine and State medicine, and sees in them the first step towards a regimentation of utilities, of industries, of finance and eventually of labour itself. Louis H. Bauer, a member of the Board of Trustees of the American Medical Association, names four elemental factors as being responsible for the present problem. These are the improper distribution of doctors, the lack of proper diagnostic facilities in certain areas, the cost of illness, and a general economic factor. These have of course all been named over and over again in Australian discussions. In 1945 the American Medical Association adopted a fourteen-point programme. Some of the items or objectives in this programme (not those dealing with insurance) are so reminiscent of our own Federal Council's policy that we reproduce them in full.

1. Sustained production leading to better living conditions with improved housing, nutrition and sanitation, which are fundamental to good health.

2. An extended programme of disease prevention with the development or extension of organizations for public health service so that every part of our country will have such service, as rapidly as adequate personnel can be trained.

3. Increased hospitalization insurance on a voluntary basis.

4. The development in or extension to all localities of voluntary sickness insurance plans and provision for the extension of these plans to the needy under the principles already established by the American Medical Association.

5. The provision of hospitalization and medical care to the indigent by local authorities under voluntary hospital and sickness insurance plans.

6. A survey of each State by qualified individuals and agencies to establish the need for additional medical care.

7. Federal aid to States where definite need is demonstrated, to be administered by the proper local agencies of the States involved with the help and advice of the medical profession.

8. Extension of information on these plans to all the people with recognition that such voluntary programmes need not involve increased taxation.

9. A continuous survey of all voluntary plans for hospitalization and illness to determine their adequacy in meeting needs and maintaining continuous improvement in quality of medical service.

10. Discharge of physicians from the armed services as rapidly as is consistent with the war effort in order to facilitate redistribution and relocation of physicians in areas needing physicians.

11. Increased availability of medical education to young men and women to provide a greater number of physicians for rural areas.

12. Postponement of consideration of revolutionary changes while 60,000 medical men are in the service voluntarily and while 12,000,000 men and women are in uniform to preserve the American democratic system of government.

13. Adoption of Federal legislation to provide for adjustments in draft regulation which will permit students to prepare for and continue the study of medicine.

14. Study of post-war medical personnel requirements with special reference to the needs of the veterans' hospitals, the regular army, navy and United States Public Health Service.

Bauer points out that item 12 is already somewhat obsolete, but that does not matter; the inclusion of the item shows that in Australia and America the organized members of the profession have been thinking on similar lines.

The final item in the American discussions to which we would draw attention is an address by Roger I. Lee, President of the American Medical Association. He asks in the title of his address: "What is Adequate Medical Care?" We have no need to follow him in his attempt to answer this question. More important is his conclusion that constant and continuing studies of the changing conditions in medical science and social standards are necessary. Clearly what is adequate today may not be adequate tomorrow. This means that our standards and our plan of campaign must be continually under review. From this it follows that plans are of no use unless we act upon them. While the concerted wisdom of the Australian Branches is reflected in the policy of the Federal Council, this policy must be constantly reviewed and must also be the basis for constant action. Even if the Commonwealth Government does not immediately try to set up a medical service according to its own ideas, the medical profession must not slacken in its efforts to bring about the "improvements" which it has declared that it desires. There is no reason why the medical profession in the Commonwealth should not be an example to practitioners in other countries.

Current Comment.

TWO CAUSES OF PAIN IN THE RIGHT ILIAC FOSSA.

DETERMINATION of the cause of a pain on the right side of the lower part of the abdomen is an important and often not an easy matter. There have recently appeared discussions of two conditions which cause such pain, but which perhaps are not thought of often enough. Ian Aird[1] describes acute non-specific mesenteric lymphadenitis and James A. Greene[2] writes of the syndrome of psoas myositis and fibrositis.

Aird writes from Edinburgh, but his description of non-specific mesenteric adenitis "as one of the commonest causes of acute abdominal pain in children and as an occasional puzzling affection in adults" is equally true of Australia as he indicates that it is of Scotland. The glands most often affected are the juxta-intestinal group of the mesenteric glands, lying along the mesenteric attachment of the lowest few feet of ileum. Later the intermediate and terminal mesenteric glands along the ileal arteries and on the main superior mesenteric trunk become involved. Sometimes the caecal and ileo-colic groups of glands are similarly affected. The patients are usually children beyond the age of infancy. The outstanding symptom is severe colicky abdominal pain, most intensely felt in the lower part of the abdomen on the right side, but not precisely localized by the patient. Nausea and vomiting may occur. Recurrent attacks are common. The temperature is often of the order of 101° F., and there is a neutrophile leucocytosis. The abdominal tenderness is higher and more medial than is usual in appendicitis, and the site of maximum tenderness often shifts to the left, perhaps even beyond the mid-line, if the patient turns from the supine to the left lateral position. The overlying muscles have increased tone, but relax under steady pressure. The enlarged glands can seldom be palpated. There is sometimes evidence of a recent or accompanying upper respiratory tract infection.

Acute appendicitis is the condition most likely to be confused with acute mesenteric adenitis. We agree with Aird that distinction is usually possible, but most clinicians have been accustomed, in making a distinction, to emphasize the disproportionate temperature and the frequent presence of a throat infection rather than the abdominal signs. Aird writes: "The sharp spasms of colic with complete or almost complete freedom from pain between them, the rolling and kicking of the child in an attack, the high and medial situation of the tenderness, the absence (if it is absent) of rebound tenderness even at the point of maximum tenderness, the presence (if it is present) of shifting tenderness, the relaxation under steady pressure of apparently rigid muscles, the history of previous typical recurrent attacks if these have occurred, all inspire confidence in a negative diagnosis of 'this is not appendicitis'." It is true that this clinical picture is more suggestive of mesenteric adenitis than of appendicitis, but acute appendicitis in the child, particularly the young child, is often an atypical and treacherous complaint, and it is a comfort that there is usually further evidence that helps to exclude it. One very valuable further sign is the temperature. Appendicitis does not cause a high temperature until it advances to peritonitis, at least local, or abscess formation. Mesenteric adenitis usually does. If the temperature is above 100° F. and it is not apparent that inflammation has spread beyond the appendix, then appendicitis will rarely be the underlying illness. The obscurity of an inflamed appendix lying in the pelvis, of course, must not be forgotten. An equally valuable fact in our experience is that mesenteric adenitis is very often, indeed we think usually, accompanied or preceded by an acute upper respiratory infection, a reddened naso-pharynx with often some post-nasal mucopurulent discharge. Aird admits that this is a "not unusual concomitant of non-specific mesenteric adenitis, but neither its presence nor its absence is highly significant, for in more than 70% of my cases pharyngitis has not been detected, and acute pharyngitis sometimes closely precedes acute appendicitis". We should have been inclined rather to the view of Olney and Adams, quoted by Aird, who regarded mesenteric adenitis as "a local manifestation of a generalized condition from upper respiratory tract infection". A confident diagnosis of acute mesenteric adenitis can often be made, but if after consideration of all these factors appendicitis cannot be excluded with assurance, then in the interests of safety operation may well be the right course.

Psoas myositis and fibrositis, described by Greene, may cause pain in either or both lower quadrants of the abdomen. It may persist for days or for even as long as years. It is increased by activity that involves use of the ilio-psoas muscles, and is revealed by tenderness along these muscles and pain on contracting or stretching them. Diagnosis is usually easy enough if the condition is kept in mind. Irritation of the psoas may, of course, result from inflammation of organs adjacent to it, particularly the appendix and urinary tract, but occasionally the spine, bowel or the female pelvic organs. Disease of these structures must be excluded before a simple myositis or fibrositis is accepted as the cause of the pain. Many incorrect diagnoses were made and ineffective treatments given before the condition was recognized in the patients reported by Greene, errors that in most cases would not have occurred had the physician thought of myositis.

[1] *British Medical Journal*, November 17, 1945.
[2] *Annals of Internal Medicine*, July, 1945.

EXCRETION OF IRON.

THE usefulness of the administration of iron by the mouth and the whole question of iron metabolism aroused one of the most protracted debates in the history of modern medicine. At one time iron in several extra-pharmacopœial as well as intrapharmacopœial preparations was given for a long list of pathological conditions from chlorosis to erysipelas. Then came a reaction and a denial that the iron admitted by the mouth ever reached the blood stream; it acted merely as an antiseptic checking the formation of products of protein putrefaction in the large intestine. This latter extreme view had to be abandoned when absorption of iron by the intestinal mucosa was definitely shown to occur. Experimental investigations on iron metabolism have always been beset with impediments; in the first place analysis is difficult, as so many reagents contain iron as an impurity; in the second place iron sulphide, formed in the intestine, is apt to cling to the mucosa and to be discarded very slowly. Again the kidneys are capable of some elimination of iron and the amount in the day's urine can show some variation—within narrow limits, it must be conceded. A. G. Little, junior, M. H. Power and E. G. Wakefield, of Rochester, Minnesota, have investigated the capability of the human being to eliminate excess iron from the blood.[1] Their method was to inject intravenously ferrous ascorbate which has been proved to be one of the safest preparations for this purpose. Let it be remembered that a distinguished pharmacologist once declared that inorganic iron is as toxic as inorganic arsenic when injected directly into the circulation—an exaggeration, perhaps, but a warning to the adventurously inclined in therapeutics. It was found that intravenous injection of relatively large amounts of iron caused no detectable increase of iron in the fæces. This result supports the conclusion of R. A. McCance and E. W. Widdowson that the body possesses no mechanism for the removal of superfluous iron.[2] No hint is given by these authors regarding the fate of the injected iron; one assumes that the reticulo-endothelial system will once more be tacitly regarded as the great impounding agent. What has emerged from other investigations conducted with "tagged" iron is that the intestine has a delicately adjusted capacity to absorb this metal when such is wanted; if the body stores are ample, then absorption automatically ceases.

[1] *Annals of Internal Medicine*, October, 1945, page 627.
[2] *The Lancet*, Volume II, 1937, page 680.

Abstracts from Medical Literature.

OPHTHALMOLOGY.

Perforating Injuries.

A. C. SNELL (*American Journal of Ophthalmology*, March, 1945) has published a study of 172 cases of perforating ocular injury and has tried to assess the factors influencing prognosis. Age has very little bearing on the outcome, whereas the influence of the agent causing the injury appears to be proportional to its sharpness. Injuries caused by broken glass have the best prognosis, blunt objects and gunshot wounds the worst. As to length of laceration, most eyes in which the length exceeds nine millimetres were excised, but no correlation was demonstrated between the length of laceration and the percentage of satisfactory recoveries. The final visual acuity varies proportionally with the length of the wound. The location of the wound does not appear to have much effect on the average of successful recoveries. Cornea, ciliary region and sclera all healed with about the same effect on vision. An exception was double perforation in which the results were bad. Prolapse of iris or ciliary body occurred 91 times; it did not affect the incidence of infection or the healing. Lens damage was disastrous, especially when associated with uveal injury. Of eyes in which the lens remained intact, 75% healed well and useful vision resulted. Of eyes in which the lens was punctured or ruptured, 45% were ultimately enucleated and in only 29% did any sight remain. The incidence of infection increased fourfold in the presence of free lens protein apart from the constant presence of *iritis phako-anaphylactica*. Fifteen eyes had severe intraocular hæmorrhage, all with bad results. In some the hæmorrhage was expulsive, in others it excited an inflammatory response which is always alarming and may give rise to a secondary hæmorrhage. In a few the blood became organized, leading to the formation of cyclitic membranes or vitreous bands and eventually *phthisis bulbi*. Intraocular hæmorrhage is frequently responsible for the quiet passing into phthisis of many injured eyes whose original appearance encouraged optimism. Infection occurred in 15%, and in all but four cases the eye was a total loss. Sulphonamide drugs given as a prophylactic measure reduced the incidence of infection, but hardly affected the result when once infection was established. Two cases of sympathetic ophthalmia occurred, both after quiet injuries at the limbus. In one case sulphadiazine had been given. Prompt recovery follows removal of the exciting eye.

Bilateral Retinoblastoma.

H. MARTIN AND A. B. REESE (*Archives of Ophthalmology*, June, 1945) report twenty-four cases of bilateral retinoblastoma. The principles of treatment were: first, the enucleation of the eye with the more advanced involvement, and second, the treatment by fractional X-radiation of the remaining eye in an attempt to conserve vision. Retinoblastoma is a fairly radiosensitive tumour. It can be completely devitalized by a dose of radiation well tolerated by the skin and underlying structures. There would therefore be little difficulty in sterilizing such a tumour except for the fact that it is not readily accessible and lies close to the radiosensitive ciliary body, lens and cornea. As a matter of fact the anterior chamber of the eye is so sensitive that it is irreparably injured by even a part of the dose necessary to destroy the tumour. Retinoblastoma is always congenital, manifesting itself later because of an increase in size. It is bilateral in more than 25% of cases. The involvement of the second eye is due not to extension across the chiasma, but to the multiple origin of the growth. The second growth is frequently missed owing to its small size and a position near the equator. A special technique was used by the author: 250 kilovolts in the tube, a 1·5 millimetre copper filter and circular portals of 2·5 centimetres adapted to the nasal and temporal sides, for cross radiation. A total of about 7,500*r* was the approximate dose at each portal. The dose is given under light chloroform anæsthesia. As a rule, the smaller and flatter the growth, the more favourable for treatment. A tumour which reaches the chorioid with its rich vascular supply is uncontrollable. Some small growths vanish completely, leaving little trace—others shrink and calcify. Among the complications, atresia of the vessels follows radiation, and this leads to secondary vascularity in the form of telangiectasia. Telangiectatic blood vessels are fragile and easily ruptured by a cough or sneeze. This may lead for a while to numerous small hæmorrhages in the fundus and an erroneous impression that the growth has recurred. Glaucoma may occur from excess dosage or a leakage of rays into the anterior chamber. Two cataracts were noted. Considerable atrophy of the retina also occurred in some cases, and in some early cases overdosage led to *phthisis bulbi*. The author describes his end results. Of 24 cases one is too recent for conclusions to be drawn; in four cases treatment failed and the eye was enucleated; three patients had recurrence in the opposite orbit. In eighteen cases the condition was completely arrested, in four of these the eye became phthisical, four other patients were blind, and in a total of ten cases the vision was good.

Lysozyme.

LYSOZYME is a substance in tears described by Fleming in 1922 which has a strong lytic action on certain organisms. It is also found in nasal mucus and in saliva, but is especially abundant in egg white, tears and leucocytes. Lysozyme is a ferment. Even in high dilutions it destroys the saprophytes of the air and is therefore thought to have the function in the eye of keeping the conjunctival sac sterile. Its significance for pathogenic organisms is debatable. B. Babudieri and G. B. Bietti (*Archives of Ophthalmology*, June, 1945) have made observations on pathogenic cocci with the electron microscope at magnifications of 18,000 to 19,400. In the first phase there is rarefaction of the bacterial protoplasm. In the second phase fragmentation and swelling occur and in the third total plasmolysis, the envelope alone remaining, filled with amorphous material. Lysis produced by bacteriophagia differs in that the envelope is surrounded by bacteriophage bodies, though in this process also the envelope remains intact. Sulphonamide compounds, on the other hand, cause dihiscence of the bacterial membrane with escape of altered protoplasm.

Strabismus Treated Orthoptically.

R. U. GILLAN (*The British Journal of Ophthalmology*, August, 1945) had one hundred and fifty patients with concomitant squint. Fifty of these were ordered glasses and were kept on the waiting list as controls. In no case was there any improvement. In some instances the condition became worse. One hundred were treated by orthoptic means, sixty-three by that alone, and thirty-seven by orthoptics *plus* operation. Of the eyes of sixty-three treated by orthoptics alone, thirty-six became straight and developed good steroscopic vision. The prerequisites of cure were the existence of simultaneous perception and fusion and a squint of less than 25°, preferably about 10°. Of thirty-seven patients treated by orthoptics *plus* operation, the eyes of twenty-three became straight and seventeen developed some stereoscopic vision. Stereoscopic vision is an important factor in the maintenance of orthopsis when operation has been performed. The eyes of fourteen patients with an average original deviation of 35° did not become straight. To sum up, in the whole series of one hundred patients the eyes of 45% became straight and stereoscopic, the eyes of 26% became straight without full stereopsis, and 29% were failures. This should be contrasted with the 100% failure among the controls.

Marginal Blepharitis.

M. E. FLOREY, A. M. MCFARLAN AND IDA MANN (*The British Journal of Ophthalmology*, July, 1945) mix 800 Oxford units of penicillin with a little water and beat this up with sterile "Vaseline" under aseptic conditions; the whole making one gramme of ointment. With this they have treated 48 patients suffering from marginal blepharitis; 30 of the cases were ulcerative, 17 were squamous and one was complicated by corneal ulcers. Duration of the symptoms varied from a month to fifteen years, but as only five patients had a history of less than two months, in the bulk of the cases the condition may be considered as having reached a chronic stage. In all but seven cases bacteriological examinations were made before treatment. In 39 cases *Staphylococcus aureus* (coagulase positive) was grown and in two *Staphylococcus albus* (coagulase negative). The patients treated themselves by applying the ointment to their lids and rubbing it in. Thirty-six of the patients applied the treatment regularly three or four times a day for as long as was considered necessary, three to ten weeks. Recovery took place in all of these without any adjuvant treatment other than the epilation of a few lashes. Bacteriological results were found to be closely associated with clinical signs. Clinical cure was invariably succeeded by failure to obtain a culture. A year later two-thirds of the patients had had no recurrence, despite the fact that concomitant conditions such as seborrhœa had been deliberately neglected.

Insulin Treatment of Corneal Ulcers.

T. R. AYNSLEY (*The British Journal of Ophthalmology*, July, 1945) reports five cases of indolent corneal ulcer which resisted all of the usual methods of treatment. The first patient had a right facial paralysis with chronic conjunctivitis and a large ulcer. Insulin drops were instilled, an excessive proliferation of epithelium occurred, and the ulcer healed in a week. In the second case severe burns made apposition of the lids impossible. Insulin was injected and the ulcer healed in six days. The third patient was a child with pink disease in very bad general condition. Three units of insulin were injected daily and healing occurred in four days. The fourth patient had chronic superficial ulceration of the cornea for ten years. An excavating ulcer near the centre of the pupil healed in three weeks with excessive proliferation of epithelium. The fifth patient was a man with severe *herpes frontalis* and a large hypopyon ulcer. Insulin injections were given and the ulcer healed in four days. Regarding the action of insulin in these cases, three possibilities suggest themselves. Insulin may improve the patient's nutrition, it may remove some factor necessary for germ metabolism, or it may increase the rate of epithelial proliferation.

OTO-RHINO-LARYNGOLOGY.

Lateral Sinus Thrombophlebitis.

H. ROSENWASSER (*Archives of Otolaryngology*, February, 1945) has made an analysis of one hundred cases of thrombophlebitis of the lateral sinus in which operation was performed. The condition occurred oftener in the five-year age period from six to ten years than in any subsequent period of life. Sixty-two of the hundred patients were males. In approximately 80% of the cases blood cultures were obtained—β hæmolytic streptococcus in 68 cases, an anaerobic hæmolytic streptococcus in five, pneumococcus type III in three, *Bacillus proteus* in two, *Staphylococcus aureus* in one, and *Bacillus pyocyaneus* in one case. After obliterative operation and ligation of the jugular vein, blood cultures were still obtained in most cases for one week and occasionally for a longer period. In some a culture was obtained from the blood only after operation on the sinus and ligation of the internal jugular vein. In several cases revision of operation on the sinus or its tributaries revealed additional foci. In 22 cases the greatest expression of disease was found in the jugular bulb. The insidious behaviour of pneumococcus type III with its tendency merely to be held in check by chemotherapy, yet its proneness to regain activity and to have increased its invasive ability, was demonstrated in several cases. Chills occurred before operation in 63% of cases. The absence of chills cannot be said to militate against a diagnosis of lateral sinus infection. In this series there was a relatively high incidence of chills in children. Metastases were noted clinically in 21 of the 100 cases before obliterative operation on the sinus; in 22 cases there were symptoms of metastasis after operation. In 13 of these 25 cases the evidence indicated that the metastases did develop after operation, although it is thought that the actual infection of remote parts occurs through bacteria liberated into the blood stream before operation. The occurrence of such late metastases is not accepted as an argument against the value of ligation of the jugular vein, but may call for a revision of the sino-jugular surgery. Metastases in the joints were more frequently periarticular than intraarticular. In children the hip joint seemed to be the site of predilection. Vague signs suggesting pneumonitis were observed in nine instances. Changes in the ocular fundus varying from hyperæmia to well-marked papillœdema were noted before obliterative operation in 24 patients. After obliteration and ligation definite papillœdema was observed in 23 cases, and in many instances a definite advance in the degree of papillœdema was noted for several days after operation. There was no significant correlation between the changes in the fundus and the condition of the patient or the severity of the otitic sepsis. Death occurred in 27 of the 100 cases, the mortality being strikingly greater among patients past thirty than among children. In several instances cerebral abscess and meningitis were the cause of death rather than the sinus thrombosis and bacteriæmia. Nineteen patients of the series were given sulphonamide chemotherapy post-operatively. The danger of the masking of symptoms and the necessity for extensive and thorough surgical drainage of all foci of infection in addition to sulphonamide therapy are emphasized. When such a course is adopted the influence of chemotherapy on the treatment of bacteriæmia is great. While the mortality appears to be greater the longer the blood infection has been in existence, it is agreed that it is better when necessary first to institute supportive measures until improvement in the general condition of the patient is evident.

Sinusitis and Penicillin.

I. J. HAUSER AND W. P. WORK (*Archives of Otolaryngology*, March, 1945) have treated with penicillin 46 patients suffering from infection in or about the nose and sinuses. Of these, 11 were treated by irrigation of the maxillary antra with solutions of penicillin, and the remaining 35 were treated by intramuscular injections of penicillin either alone or in conjunction with radical surgical opening of the sinuses. For instillation into the antra the solutions contained 250 to 1,000 units of penicillin per millilitre, 5·0 millilitres of solution being instilled into each antrum. For parenteral treatment the intramuscular route was always used, doses of 10,000 units being tried at first. In later cases doses of 15,000 units were used, and later still 20,000 units every three hours has been the dose of choice. The series of cases included three of allergic rhinitis with superimposed infection. In each instance there occurred complete disappearance of pus, with recession of polypoid tissue, but some persistence of mucoid discharge. In four cases of orbital cellulitis there was rapid subsidence of all signs of infection. In three cases of suppurative sinusitis secondary to shrapnel wounds, once an intranasal plastic operation was done and intramuscular administration of penicillin was continued, complete disappearance of all suppuration of the sinuses occurred. In nine cases penicillin treatment was combined with radical operation on the antra. Exudate disappeared from the nose and sinuses within a few days after the penicillin treatment was started. In three instances administration of penicillin was started a day or two before operation and continued afterwards. In these cases there was only minimal post-operative reaction and complete resolution was rapid. In nine cases a radical operation was performed on the frontal and ethmoid sinuses. In three of these intracranial complications were present, and penicillin treatment was adopted before and after operation, and with sulphadiazine as well. Recovery was complete in each instance. At first the authors thought that early striking successes achieved in cases in which only shortly established inflammation was present, indicated the possibility that penicillin alone might effect a cure in cases of chronic suppurative sinusitis, especially if of short duration. It is still their practice to try the effect of adequate amounts of penicillin intramuscularly before considering surgical intervention. In cases of long-standing infection adequate surgical treatment is found to be necessary. When penicillin treatment is combined with such surgical measures, rapid cure, or at least results far better than previously obtainable, have been achieved. Every patient who is to have radical surgical treatment is now given penicillin for two days prior to operation and injections are continued post-operatively for as long as necessary.

Penicillin Therapy by Inhalation.

E. W. HAGENS, MARY KARP AND C. J. FARMER (*Archives of Otolaryngology*, May, 1945), describing an inhalation method for penicillin therapy, state that a solution containing 8,000 units of penicillin per millilitre dissolved in equal parts of distilled water and peppermint water (the latter added to mask the musty odour of penicillin) is aerosolized by blowing oxygen through a de Vilbiss nebulizer. The rate of flow is adjusted to nebulize 1·5 millilitres (12,000 units) in five minutes, which is the period of each treatment. The patient inhales the vapour through a mask equipped with a rebreathing bag. Treatments were given sixteen times daily over a period of seven to ten days, the daily dose approximating 192,000 units. Twenty-two cases in which patients suffering from pulmonary disease were treated by penicillin inhalation therapy are reported. These comprise ten suffering from bronchiectasis, five suffering from pneumonia, one from lung abscess and two from sinusitis and pharyngitis. Bacterial flora were recorded before and during treatment. It was observed that Gram-positive bacteria quickly disappeared, but Gram-negative organisms persisted and sometimes appeared, although absent from original cultures. In assays of urine up to 20% of the penicillin was recovered. Blood serum levels were found to be variable. Some temporary improvement in bronchiectasis was noted, but the condition relapsed shortly after the therapy was discontinued. The condition of all of the five patients with pneumonia was improved, although sulphonamides had previously failed to confer any benefit. One patient with lung abscess recovered.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on November 22, 1945, at Broughton Hall Psychiatric Clinic, Sydney. The meeting took the form of a series of clinical demonstrations arranged by Dr. S. Evan Jones, the Medical Superintendent.

Neurosyphilis.

Dementia Paralytica.

The first patient shown was a male, aged forty-three years, whose symptoms had first presented themselves nine months earlier; at that time he had a "turn", during which he was unconscious for twenty minutes, and he had a similar attack a few days later. About this time it was noticed that his speech was slurred. During the two months prior to the meeting he had had a number of seizures in which he complained of feeling giddy and turned pale, and his consciousness became clouded; he had no convulsions, and for some time afterwards he was confused. After an attack which occurred two weeks prior to the meeting, his right arm and leg felt stiff and weak. He had had auditory hallucinations and had talked in an extravagant manner. He exhibited considerable mental deterioration and at times he was confused.

Examination revealed gross tremor of the facial muscles and tongue; the speech was slurred, the tendon responses were brisk and the abdominal responses were hyperactive. This was said to be a common finding in neurosyphilis. The pupils were slightly unequal in size and rather small. The right pupil was sluggish in its reaction to light, and both pupils responded briskly to accommodation. The blood serum reacted to the Wassermann test. The cerebro-spinal fluid also reacted to the Wassermann test; the cells in the fluid numbered 18 per cubic millimetre, the globulin content was increased, and the gold sol test produced the following curve: 3455432100.

The comment was made that this condition was a commonplace type of *dementia paralytica*; it was well advanced, as was indicated by the mental deterioration and by the occurrence of epileptiform and apoplectiform seizures, which did not as a rule occur until the second stage. The patient had been receiving injection treatment for twelve months. It was not sufficiently recognized that paretic types of neurosyphilis would not respond to arsenical therapy alone; some form of pyrexial therapy was essential and should be given as soon as the diagnosis was made. The patient had now finished a course of "inductotherm" pyrexia and was being given weekly injections of tryparsamide. Even if the disease process had been arrested, as it should have been, the ultimate result would not be good, for there was certain to be a residual dementia of considerable degree.

The next patient was a female, aged thirteen years, suffering from congenital *dementia paralytica*, whose mother had died in 1936 of *dementia paralytica*; she had had six children. In Table I are set out the responses in 1936 of the six children's serum and cerebro-spinal fluid to tests for syphilis. The father's blood serum and cerebro-spinal fluid failed to react to tests for syphilis, and he had no clinical signs of syphilis. The children who gave positive reactions were treated, D. being given a course of malaria.

TABLE I.

Patient.	Sex.	Age in 1936. (Years.)	Response to Tests for Syphilis.	
			Serum.	Cerebro-spinal Fluid.
M. ..	Female.	17	Positive.	Negative.
T. ..	Male.	15	Negative.*	Negative.
M. ..	Female.	12	Negative.	Negative.
J. ..	Male.	11	Positive.	Negative.
D. ..	Male.	8	Positive.	Positive.
E. ..	Female.	3	Positive.	Negative.

In 1945, the condition of the children was the following. M., aged twenty-five years, was married and had one child; her responses to tests for syphilis were negative. T., aged twenty-three years, gave negative responses to tests for syphilis. M., aged twenty years, failed to yield reactions to tests for syphilis. In the case of J., negative results had been obtained for two years. D. was admitted to hospital for investigation in April, 1945; he was aged sixteen years, was mentally defective and was subject to epileptiform seizures; his response to tests for syphilis was negative. This boy presented the end-result of arrested neurosyphilis of the paretic type. E., the patient under consideration, although of good intelligence, was morally irresponsible; she stole, lied and misconducted herself. Her blood serum did not react to the Wassermann test; her cerebro-spinal fluid reacted to the Wassermann test and contained an increased amount of globulin, while the gold sol test produced the following curve: 5555543100.

Dr. Evan Jones said that this was a case of congenital neurosyphilis of the paretic type. It was noteworthy how arbitrarily the maternal syphilis was transmitted to the children, the manner suggesting that the transmission was via the germinal cells. Congenital paresis responded to pyrexial treatment in the same way as acquired states; the end-results were usually bad, because treatment was instituted at a late stage in the evolution of the disease.

The next patient was a male, aged forty-one years, for whom no reliable history was available. He was suffering from the simple dementia type of *dementia paralytica*. The patient stated that he had worked until ten weeks previously, and that he was regarded by his mates as being "punch-drunk" (he had been a professional pugilist). He had *lues* fourteen years previously, for which he was treated for six weeks with injections into his arms and buttocks. On his admission to hospital, he exhibited general mental deterioration and was dull and stupid; his memory and orientation were defective. However, he showed no delusional symptoms or any emotional disorder. He looked debilitated and prematurely aged. The pupils were slightly unequal; the left was irregular in outline, and both were sluggish in their reactions to light. Speech was slow and stumbling, but was not noticeably slurred. Slight tremor of the tongue was present. The tendon responses were active and the abdominal reflexes were brisk. The blood serum yielded a "partial positive" reaction to the Wassermann test. The cerebro-spinal fluid reacted to the Wassermann test; it contained eleven cells per cubic millimetre and an increased amount of globulin; the gold sol test produced the following curve: 5543321100.

Dr. Evan Jones said that this case conformed to the common type of *dementia paralytica*; the "classical" form, with expansive delusions, euphoria and hyperactivity, was relatively rare. Dementia was the sign of destruction of cortical cells in the frontal regions, while delusions and other psychotic symptoms represented release phenomena. The prognosis in the case under discussion was not good; although the disease would in all probability be arrested, considerable irreversible cortical damage had already been done, as shown by the dementia.

Neurosyphilis: Tabetic Form with Optic Atrophy.

A male patient, aged twenty-nine years, in March, 1943, lost the use of his left hand and foot and was unconscious for two days; the attack passed off in a few days. He had other similar episodes in June, 1943, and January, 1944. He had become forgetful, his vision had failed and his legs gave way under him. He was admitted to hospital in February, 1944, and was given malaria therapy with considerable general improvement. He had received a good deal of arsenical and bismuth treatment before his admission. At that time he presented a tabetic type of neurosyphilis, with absence of knee jerks, ataxia of extremities and considerable optic atrophy. In view of the toxic effect in the optic nerves attributed to tryparsamide, he was given a course of "Marpharsen" injections. Since the syphilitic process was found by tests to be still active, he had been readmitted to hospital for a further course of pyrexial therapy. As he was almost completely blind, this would be followed up with a course of tryparsamide. In February, 1944, the serum reacted to the Wassermann test; the cerebro-spinal fluid reacted to the Wassermann test, and the gold sol test produced the following curve: 5544311000. In November, 1945, the serum and the cerebro-spinal fluid reacted to the Wassermann test, and the gold sol test produced the following curve: 4444321100.

Dr. Evan Jones said that, although no serological response to treatment had occurred, there had been considerable clinical improvement except with respect to the optic atrophy. Tabetic optic atrophy was often of rapid evolution, and once established, it was an irreversible condition. The patient's disks were in an advanced stage of atrophy. His other physical signs were absence of knee jerks and ataxia.

Another patient shown was a male, aged thirty-eight years, who two years previously had noticed that his eyesight

was failing. His serum had been found to react to the Wassermann test. He was not aware that he had ever suffered from syphilis. He rapidly lost vision in his right eye, and that of his left eye was also defective; but he had no other symptoms. On his admission to hospital, he was found to have internal strabismus of the right eye, the vision of which had been almost completely lost. The pupils were unequal and inactive to light. Slight Rombergism was present, but no obvious loss of joint-muscle sensation could be detected. The knee jerks were absent, and the plantar response was extensor in type on the right side. The abdominal reflexes were hyperactive. The serum failed to react to the Wassermann test. The cerebro-spinal fluid reacted slightly to the Wassermann test; the cell content of the fluid was five per cubic millimetre, the globulin content was slightly increased, and the gold sol test produced the following curve: 0121100000. The patient had an old tuberculous lesion of the apex of the right lung, without signs of activity.

Dr. Evan Jones said that sometimes failing vision was the first warning of neurosyphilis, and often the evolution was rapid and optic atrophy quickly developed. The treatment of this complication of neurosyphilis was unsatisfactory; arsenicals were contraindicated because of their neurotoxic properties, and probably the best therapeutic measure was pyrexia. The syndrome of the patient under discussion was of tabetic type, but there was evidence of pyramidal involvement, the basis of which was most likely lateral sclerosis of the cord. Pulmonary tuberculosis was a contraindication to pyrexial therapy; but in view of the absence of signs of activity, it was intended to give the patient a therapeutic course of malaria.

Neurosyphilis: Tabo-Paretic Type.

The second patient shown was a male, aged thirty-six years. His first sign of trouble was a convulsive seizure early in 1943, although during medical examination on his enlistment it had been noticed that his knee jerks were diminished. Since the first attack he had had about ten others at varying intervals. In the seizures he called out, lost consciousness and had convulsions, coming to himself in about twenty minutes. He sometimes bit his lips and had injured himself by falling during the seizures. Two months prior to the meeting his speech became slow and hesitant, although not slurred. When he was admitted to hospital he manifested some emotional instability, but no other mental symptoms. Examination showed the pupils to be small, equal, regular and active; the knee jerks were absent and the abdominal reflexes were hyperactive. The blood serum failed to react to the Wassermann test. The cerebro-spinal fluid gave a partial positive reaction to the Wassermann test; cells numbered eleven per cubic millimetre, the globulin content was slightly increased, and the gold sol curve was 4312100000.

Dr. Evan Jones said that the absence of knee jerks suggested a tabetic type of lesion, but very little ataxia was present and there was no evidence of mental deterioration; thus the characteristic sign of *dementia paralytica* was lacking, although the occurrence of epileptiform seizures was highly suggestive of that type of neurosyphilis. The pathological process was probably not very active; this was suggested by the absence of more striking paretic or tabetic signs and by the rather low-grade positive serological findings. The man was started on a course of electropyrexia, but proved to be non-cooperative. He was subsequently inoculated with malaria parasites, and at the time of the meeting he had had four pyrexial attacks. The prognosis for the arrest of the disease was good, and probably his convulsive seizures would also be stopped.

Comment.

Discussing the diagnosis and treatment of neurosyphilis, Dr. Evan Jones said that symptoms of syphilitic involvement of the central nervous system fell into four main syndromes: meningitic syphilis, cerebral or vascular syphilis, paresis and tabes. Although at times the clinical pictures were clear-cut, more usually they were mixed, so that complicated or transitional forms were seen. Acute syphilitic meningitis sometimes developed in the secondary stage; the symptoms were similar to those of cerebro-spinal meningitis, and usually the disease was rapidly fatal. More commonly the meningitic reaction was subacute or chronic and was associated particularly with the vascular and paretic syndromes. Cerebral syphilis was properly a disease of blood vessels, the cerebral arteries sharing in a common syphilitic endarteritis. Symptoms were caused by thrombosis, and the usual picture was hemiplegia. It was possible that too intensive treatment in the early stages of the infection might cause an excessive endothelial proliferation leading to occlusion. This was one form of the so-called Herxheimer reaction. Paresis was a cerebral parenchymatous form of neurosyphilis; it was a progressive disease with a rapidly fatal termination if left untreated. In tabes the gravamen of the pathological process was in the posterior columns of the spinal cord.

Dr. Evan Jones went on to say that while vascular syphilis was sometimes catastrophic in its onset, it usually showed no progressive tendency. Unless it was complicated by paretic changes, it responded to ordinary antisyphilitic treatment. If there were no accompanying meningitic changes, the cerebro-spinal fluid might fail to react to tests for syphilis. The distinction between tabes and paresis was readily made on clinical findings. Sometimes mixed forms were present, constituting the so-called tabo-paresis. Sooner or later in the course of parenchymatous and meningitic forms of neurosyphilis the cerebro-spinal fluid would react to the Wassermann and colloidal gold tests and would contain an increased number of lymphocytes and an increased amount of protein. A high pleocytosis pointed to an active meningitic process. Although significance had been attributed to the type of gold sol reaction in the differentiation of meningitic, paretic and tabetic syndromes, experience at Broughton Hall did not bear this out. The real value of the cerebro-spinal fluid findings, apart from the aid they gave in the diagnosis of neurosyphilis, was as an indication of the activity of the pathological process; for example, high pleocytosis, an increased globulin content and a gold sol curve in the higher numbers pointed to great activity of the disease process. As the disease responded to treatment, there occurred first of all a fall in the cell content of the cerebro-spinal fluid, and then a fall in the protein content of the fluid, and later again lower numbers appeared in the gold sol curve. Last of all the Wassermann test ceased to produce a reaction. In summary, the diagnosis of the form of neurosyphilis was made on the clinical findings and the degree of activity of the process as revealed by investigation of the cerebro-spinal fluid.

Dr. Evan Jones then said that cerebral syphilis and tabes would respond to ordinary antisyphilitic treatment, and both at times burned themselves out. Paresis, on the other hand, progressed to a fatal termination, and would not respond to intravenous medication alone. The introduction of malarial therapy was based on the experience that paretics who had intercurrent pyrexial infections sometimes had remissions. Although there was no general agreement as to the mode of action of malaria, it was assumed that the beneficial effect was produced by the pyrexia. This suggested the use of other means of inducing pyrexia. Neymann in the United States of America had first used diathermy and later short-wave induction for this purpose. The combination of pyrexial treatment with the intravenous administration of arsenic proved to be completely effective in arresting the course of paresis. Dr. Evan Jones advised its use also in tabes as a valuable if not essential adjuvant to metallotherapy.

Disseminated Sclerosis.

The next patient had been referred to Broughton Hall on account of an attack in which he lost control of his arms and legs and had blurred vision; he had had three similar attacks. The gold sol test had produced the following curve: 5555543100; but otherwise serilogical findings were negative, and it was suggested that he was a suitable subject for pyrexial treatment. The patient said that in 1937 he had had similar symptoms. His condition had improved, and he had worked as a seaman until February, 1944, when he was incapacitated by generalized trembling. He had lost control of his legs and his eyesight had deteriorated.

Examination revealed bilateral nystagmus, pronounced intention tremor of the upper extremities, coarse tremor of all voluntary muscles and gross ataxia of the lower extremities. His abdominal reflexes were absent. The tendon responses were hyperactive, and the plantar reflexes were extensor in type. The pupillary reactions were normal, and examination of the optic fundi revealed atrophy, the temporal halves of both disks being pearly white. His speech had a sing-song character.

Dr. Evan Jones remarked that the picture was typical of disseminated sclerosis; the age of the patient and the history of remissions were entirely consistent with that condition. There was nothing to suggest neurosyphilis other than the paretic type of curve produced by the colloidal gold test. A positive reaction to that test, however, was not conclusive proof of the presence of a syphilitic process, and it was extremely rare in known cases of neurosyphilis for the gold

sol test to produce a positive reaction when other serological findings were negative; actually in the course of treatment the response to the gold sol test became negative long before the result of the Wassermann test was reversed. Moreover, in disseminated sclerosis it was usual for some change to occur in the gold sol curve. Kinnier Wilson, for instance, stated that in 25% of cases the curve was "negative", in 25% of cases "paretic", and in 50% of cases "intermediate".

A Syndrome Resembling Disseminated Sclerosis as a Sequal to Encephalitis Lethargica.

A female patient, aged forty years, said that nine years earlier she had had an illness which began like an attack of influenza; three days after the onset she felt "dopey", and her eyelids began to droop. She became drowsy and slept for fourteen days, and during this time she had double vision. After the illness she had difficulty in walking; but this condition improved sufficiently for her to play tennis. After a few games, however, she had difficulty in moving her legs; as she expressed it, her right leg "seemed unwilling". About three years after her illness she noticed frequency of micturition. A year prior to her admission to hospital she had retention of urine for a few days; at the time of the meeting micturition was precipitate. She was constipated. She felt unsteady when she attempted to stand, and had an inclination to fall backwards.

On examination of the patient, the cranial nerve functions were found to be intact. No sensory changes could be detected. Her lower extremities were spastic, especially the right; she stood unsteadily on a wide base and walked with difficulty with the aid of a stick; her gait had a spastic character. The tendon reflexes were hyperactive, especially on the right side; ankle clonus was present on the right side, the plantar responses were both extensor in type, and the abdominal reflexes were absent. The pupillary reactions were normal. Slight nystagmus was present when the patient looked to the right. Past pointing with the right hand and foot was present. Speech seemed more deliberate than normal, and sometimes it appeared slurred. Her face was sallow and her lips were rather blue. Examination of the blood revealed that the red cells were normal and the colour index was 0·9. Neither the blood serum nor the cerebrospinal fluid reacted to the Wassermann test; the cerebrospinal fluid was normal.

Dr. Evan Jones said that from the patient's description of the symptoms, her illness at the age of thirty-one years was conclusively an attack of *encephalitis lethargica*, and on the principle of *entia non multiplicanda præter necessitatem*, her present condition was the sequel of that disease. The usual nervous complications and sequelæ of *encephalitis lethargica* were extrapyramidal, the most common syndrome being Parkinsonism. However, involvement of the pyramidal system was not unknown, and sometimes a syndrome resembling amyotrophic lateral sclerosis was seen. Souques had described as encephalitic sequelæ symptoms resembling those of disseminated sclerosis, and there was no doubt that the condition of the patient under discussion conformed to that type. Sequelæ of *encephalitis lethargica* often appeared immediately, being due presumably to damage to nerve tissues done at the time of the acute illness. Sometimes, however, the appearance of secondary symptoms was delayed, and they appeared after an interval; in this case they were attributed to a continuing infection or to secondary degeneration set up by the original disease process. Pathological evidence had been produced to show that an active, apparently infective process might continue long after the acute symptoms had subsided.

Huntington's Chorea.

The next patient shown was a female, aged fifty years, who was married and had seven children. Choreiform movements had been noticed by her friends for four years and had become much more severe during the last two years. Her mental state had altered; she had become irresponsible in many ways and was forgetful and careless in her habits, neglectful of her own needs and indifferent to her family duties. Her speech also had become indistinct. The patient's father had died of cancer at the age of sixty-two years, and was not known to be choreic; the mother was still living, and her mental state was said to be good, except that she was forgetful. The patient had one brother and four sisters. The only other member of her family who had been examined, a sister, exhibited early chorea affecting her face, head and neck; but she herself was unaware that she was affected. The patient had seven children living; no reliable information was available concerning them, except that one, aged nine years, was mentally defective and had choreic movements like the mother.

When the patient was examined in bed on her admission to hospital, she exhibited generalized choreic movements of moderate amplitude affecting her face, trunk and limbs. Her gait was unsteady and her speech was disordered by choreic interruptions of expiration. Her mental state was one of moderate dementia, her memory was defective, her interests were diminished, and insight into her condition was lacking. Dr. Evan Jones remarked that this was characteristic of the dementia of chorea; one saw patients in the advanced stages who had no realization of their defects. It had not been possible to secure evidence that the patient's father or mother was choreic; but the presence of choreic symptoms in a sister and a daughter was convincing evidence that the condition was one of hereditary chorea. It was difficult to secure precise information about the members of choreic families unless they could be examined, for the choreic and mental symptoms frequently eluded the observation of persons not familiar with the disease, and on the other hand sometimes dementia occurred without chorea; in that case, without evidence of chorea in other members of the family, a diagnosis would be impossible.

Dr. Evan Jones went on to say that amongst the degenerative nervous diseases which manifested themselves in middle life, chronic progressive chorea was one of the most striking. Usually in Australia the hereditary form described by Huntington was seen; but progressive degeneration with choreic manifestations occurred in which the hereditary character was lacking. The characteristics of Huntington's chorea were progressive mental deterioration, choreiform movements and hereditary transmission as a Mendelian dominant. Symptoms usually manifested themselves in the fourth or fifth decade, but might not appear until late in life; occasionally, on the other hand, they might appear in the first and second decades (the so-called phenomenon of anticipation). Fallacies arose, inasmuch as the records of earlier generations applied mostly to subjects who were progenitors. Since the hereditary factor was a Mendelian dominant, 50% of children of a choreic would be expected to inherit the disease, since the choreic parent was a hybrid—the result of mating of a choreic and a non-choreic. A pure-bred choreic parent would have 100% of choreic children, and this occasionally occurred in isolated communities. In the family of a patient who had recently been receiving care, the father was one of eleven children, five of whom were choreic; the patient and a sister were choreic, and they had two non-choreic siblings.

Dr. Evan Jones then said that the classical characteristics were chorea, mental decay, appearance of the disease in adult life and progressive course; but each character was subject to modification—for example, chorea might be present without dementia, or the reverse, and the onset might be in early or late life. The course might be slow or rapid. Choreic movements affected all voluntary muscles except those of the eyes. When they first appeared they were unnoticed by the patient, and if observed by friends were regarded as "fidgets"; they increased in range and extent, so that ultimately all the body musculature was in constant movement during waking hours. Speech was disturbed by disorderly interruption of respiratory movements. Dementia was an outstanding feature; the earliest signs were emotional rather than intellectual changes, the patient becoming irritable and excitable. Changes in character appeared, morality was blunted, interest decayed and the sense of responsibility failed. Memory failed, and intellect became progressively degraded.

Chronic Progressive Chorea.

A female patient, aged fifty-two years, was then shown; her father, who had died at the age of forty-seven years, had suffered from "rheumatism", her mother had died at the age of eighty-three years, and she had two surviving sisters. No members of the family had been recognized to have chorea, but information was scanty and not reliable, and the possibility of a family background of chorea could not be excluded. For seven years the patient had been "slipping"; she had become indifferent to her home responsibilities and had spent much of her time wandering aimlessly. She had become careless about her personal hygiene and clothing, and her manners had deteriorated. She had had choreic movements for two years. On her admission to hospital, she showed no gross defects of memory or orientation. She was unaware that she made any abnormal movements. Slight choreic movements were present, confined principally to the head and shoulders and the upper part of the trunk. Her speech was slurring and sometimes explosive.

Dr. Evan Jones said that the case was one of chronic progressive chorea; a family history of chorea had not been ascertained, but could not be excluded. The early symptoms were of the nature of a change in character, and it was to be noted that choreic symptoms appeared much later and were not yet pronounced. They might, in fact, readily escape notice. This patient illustrated the difficulty that occurred in the recognition by relatives of the existence of chorea in the family.

Organic Brain Disease: Possibly Cerebral Tumour.

A male patient, aged fifty-one years, had been admitted to hospital on October 29, 1945, and was said to have been well until August 15; on that day he returned from work intoxicated, had an outburst of weeping and accused his wife of immoral conduct. A few days later he attempted to throttle her. Since then he had been depressed and emotionally unstable, and had repeatedly accused his wife of carrying on with other men. He had been unable to sleep, and had been drinking to excess, and had complained of failing vision.

On his admission to hospital, he expressed ideas of his wife's infidelity and exhibited rapid changes of mood. His memory was defective. Examination revealed some weakness of movement of the left side of the face; the pupils were unequal and irregular in outline and gave a hippus type of reaction to light, and the response to accommodation was active. The knee jerks were sluggish, the right more than the left, the abdominal reflexes were active, and the plantar reflexes were flexor in type. Both optic disks were congested, and early papilloedema was possibly present. His speech had a mumbling quality, but was not slurred.

Dr. Evan Jones pointed out that a change of personality in middle life in a person previously well, integrated and stable, as in the present case, should arouse suspicion of the operation of some organic factor affecting the brain. The common organic cerebral diseases of the fifth decade were neurosyphilis, cerebral tumours and various degenerative diseases such as progressive chorea or presenile dementia. Such diseases as these often made their first appearance insidiously, with a gradual or seemingly sudden change of personality. They might be accompanied by symptoms which were usually regarded as functional, and which might suggest the diagnosis of neurasthenia or psychoneurosis. The mental symptoms of the patient under discussion were suggestive of paraphrenia, which was the most common psychosis of that time of life. Paranoid symptoms, however, were frequently seen in organic disease of the brain, as, for example, in general paralysis of the insane. Psychological symptoms suggesting an organic disease in the patient under discussion were his emotional instability, defect of memory and inadequate orientation. The physical signs were not conclusive, but would be consistent with neurosyphilis or cerebral tumour. The diagnosis of cerebral tumour was supported by the presence of early fundal changes suggestive of papilloedema and by the high pressure of the cerebro-spinal fluid (220 millimetres). The pupillary changes and diminished knee jerks, however, pointed to neurosyphilis; the high cerebro-spinal fluid pressure was not inconsistent with that condition, for high intrathecal pressures were found in the presence of a very active neurosyphilitic process. However, the diagnosis of neurosyphilis was excluded by the absence of any positive findings in the blood serum or cerebro-spinal fluid. The possibility that the condition might be secondary to chronic alcoholism should not be ignored; this would mean the discounting of the history of a sudden change of habits. Against a diagnosis of acute alcoholic psychosis was the absence of signs of peripheral neuritis and of active mental confusion.

Progressive Pontine Degeneration.

The symptoms of a female patient, aged twenty-six years, had appeared three years earlier; she then noticed for the first time weakness and stiffness in the legs and difficulty in walking, and later her back was affected. About one year prior to her admission to hospital her right eye turned in and her jaw dropped. Later her face became weak and her left eye turned in. Lastly her hearing went, and she had been totally deaf for about six months. Prior to the loss of hearing she had noises in the ears. She had attacks of palpitation. The patient said that her muscles were growing thin, but no wasting was obvious except in her face.

On examination of the patient, her sense of smell was found to be unaffected. Vision was defective in the right eye (myopia), good in the left. No papilloedema was present. The oculomotor functions were intact except for bilateral abducens paralysis. The pupillary reactions were normal. The motor functions of the fifth nerve were completely lost; the jaw fell under the action of gravity. She had weak opening movements of the jaw. Wasting of the masseter and temporal muscles was present; jaw jerks were absent. The patient complained of a numb feeling in the forehead, but she recognized light touch; sensitivity to pain was diminished over the trigeminal area on both sides. She had noticed twitching in the face and in the tongue. The facial muscles on both sides were completely paralysed, and no movements of expression were made. Mechanical irritability of the facial muscles was increased. The sense of taste was preserved. Movements of the palate were good on both sides. The tongue could be protruded with superficial tremor; some wasting was probably present, but fibrillary twitchings had not been observed. The sterno-mastoid muscles were weak and possibly somewhat atrophied; the head was ill supported and tended to fall from side to side. Phonation was good, but articulation was defective; yet speech was intelligible. Mastication was impossible, but the patient swallowed by pressing the bolus backwards between the tongue and the palate. The general sensory functions were well preserved, but the motor functions were grossly disordered; she could not stand without support, and had difficulty in sitting up. Muscular power generally was much diminished, and movements were clumsily performed and revealed dissociation of cerebellar type. The deep reflexes could be elicited, but were not exaggerated; the abdominal reflexes were present. The plantar responses were extensor in type. The blood picture was normal, and investigations of the blood serum and cerebro-spinal fluid gave negative results; the pressure of the cerebro-spinal fluid was 75 millimetres. The patient's mental state was well preserved; no dementia was present, and she had good appreciation of her illness and cooperated well within the limits imposed by her deafness.

Dr. Evan Jones said that it was necessary to explain the occurrence of bilateral lower neurone paralysis of the fifth, sixth and seventh motor nerves with early involvement of the eleventh and twelfth cranial nerves, generalized motor paresis, ataxia, bilateral pyramidal tract affection and deafness. The possibility of a tumour or tumours of the brain stem or the cerebello-pontine angles seemed to be excluded by the symmetrical distribution of the symptoms and by the absence of signs of intracranial hypertension. The early march of events, with signs of primary pyramidal tract affection followed by involvement of motor nuclei of the brain stem, was rather suggestive of amyotrophic lateral sclerosis with major incidence in the pontine region. However, the eighth nerve involvement was hardly consistent with this syndrome. The possibility of multiple neurofibromatosis should be considered; but in that case there were difficulties in explaining the pyramidal and cerebellar signs. Dr. Evan Jones was inclined to regard the condition as an anomalous ponto-cerebellar atrophy with its principal incidence in the pontine motor nuclei.

Presenile Dementia.

A female patient, aged fifty-one years, had been admitted to hospital with the history that in August, 1945, she had had a "stroke", in which her right arm was paralysed. Since that time her memory had been defective, especially for recent events, and she had shown deterioration in personal hygiene. On examination of the patient, no evidence of the recent stroke was found, nor any other focal signs of disease of the brain. The blood pressure was normal, and no abnormality was detected in the blood serum or the cerebro-spinal fluid. The striking feature was gross defect of memory for both remote and recent events. She was totally disorientated for time and place. She showed a striking tendency to confabulate, but her confabulation was stereotyped; for example, she always said that she was in Gosford, that she had arrived at 2 a.m. on the previous day, and that she was overcarried in the train.

Dr. Evan Jones said that the psychological symptoms of this patient conformed to the type of memory disturbance found in Korsakow's psychosis, in which amnesia and a tendency to confabulate were associated with peripheral neuritis, the most common ætiological factor being alcoholic excess. In the case under discussion there was no history of alcoholism and no evidence of polyneuritis. Amnesia and confabulation, however, frequently occurred in other organic cerebral affections, and often in the so-called presenile dementia or presbyophrenia. This was a progressive cerebral degeneration which manifested itself in the fifth and sixth decades. Although its ætiology was unknown, its pathology was well recognized, and on the basis of various microscopic changes certain special syndromes were differentiated—for example, Alzheimer's disease and Pick's disease; in those conditions usually disorders of the speech functions were

present, manifested by aphasia and apraxic symptoms. The patient under discussion did not yet show such signs. The "stroke" that she had had two months earlier was an apoplectiform attack, such as was common in organic disease of the brain.

Anorexia Nervosa.

An unmarried female patient, aged twenty-four years, had been admitted to hospital on July 4, 1945. She had begun to avoid her meals at the age of sixteen years, and since then she had never willingly taken adequate food. She had had much treatment and had been admitted to hospital on a number of occasions without benefit. In the course of time she had acquired great art in the evasion of feeding, and if made to take food she would regurgitate it or hold it in her mouth until an opportunity offered to dispose of it. She had achieved mastery of her family environment, and her mother had found it impossible to make her take more than a minimum quantity of food. The patient had had amenorrhœa for years. At the time of her admission to hospital her weight was four stone four pounds; she was emaciated, but no œdema was present. Her avoidance of food dated from a rude enlightenment about "the facts of life".

Dr. Evan Jones said that so-called *anorexia nervosa* was one of the forms in which conversion hysteria manifested itself. It usually occurred in adolescent girls and young women, and had its source in a variety of motives. Not infrequently the condition developed as a sequel to a voluntarily adopted slimming regimen; the hunger sensations were at first ignored and later suppressed. In other cases, the anorexia was an expression of an emotional state of disgust, which usually had its foundation in psychic aversion from sexual activities. In both groups of cases other factors were generally involved, and most patients who had persistent hysterical anorexia had poorly integrated personalities and were immature and dependent. The symptoms might be a means of achieving notice or of securing attention. With regard to treatment, no cooperation could be expected from the patient; not only was no appetite present, but there was a positive aversion from food; moreover, as a result of the long conditioning period, the stomach was physiologically incapable of accepting large quantities of food, which therefore had to be given in small amounts frequently. To stimulate real appetite insulin was given, starting with five units twice a day and working up to ten or twenty units; but the patient had to be watched for symptoms of hypoglycæmia. Vitamin B_1 was given to counter the deficiency which was usually present. Dr. Evan Jones said that these patients could be expected to use every method of deceit in their repertoire to evade food; consequently the closest observation and control over their activities were required. The patient under discussion, three months after her admission to hospital, weighed six stone five pounds, a gain of 28 pounds; but she still used various artifices to evade food, or having eaten it, to regurgitate it.

Confusional Psychosis.

A female patient, aged thirty-eight years, had been admitted to hospital on October 12, 1945; for five weeks she had been "out of her mind", and had had ideas that people were talking about her and persecuting her. On her admission to hospital she had active visual and auditory hallucinations, her memory was unreliable for remote events and more so for recent events, and she was disorientated for time. She made contradictory statements and filled gaps in her story by manifest fabrications. Her emotional state was one of irritability and apprehension; at times she seemed terrified by the expectation that she was to be tortured. She was extremely restless and moved continually about her room, disarranging her bedding. Her habits were faulty. Examination revealed that she was debilitated, and that she had paresis and tremor of the facial muscles and tremor of the tongue and hands. Her reflexes were normal, and no signs of peripheral neuritis were found. When she was admitted to hospital alcoholism was denied; but later her friends stated that she had been drinking very heavily. She continued to be extremely restless, sleepless and agitated; she frequently exhibited all the manifestations of terror, expressed delusions of persecution and had auditory and visual hallucinations.

Dr. Evan Jones said that the picture presented by this patient was an acute confusional psychosis of the Korsakow type. Although she showed slight evidence of peripheral neuritis, there was little doubt that she was suffering from toxic encephalitis. The usual treatment of insulin and thiamin injections was given, but the confusional state did not abate. After a week or two, in view of the absence of evidence of peripheral neuritis, it was considered that the primary toxic confusion had passed into a functional state, and convulsant therapy was given, with the result that the patient became lucid within a few days. It was important to remember that originally organic states might be continued as functional conditions, and that therapy had to be modified accordingly.

Another female patient, aged fifty-three years, had become depressed after the death of her son in 1944; she could not reconcile herself to her loss and gave up all interest in her home and current events. She began to drink, and was often in a muddled condition. She took sedatives in excess. When she was admitted to hospital in July, 1945, she had auditory and visual hallucinations, expressed persecution delusions, made mistakes of identity, was disorientated for time and place and related events that had not occurred. She was emotionally unstable and often irritably resistive. She exhibited no particular physical signs, other than slight hyeralgesia of the calf muscles. Her state of active confusion continued for some weeks, and she often made false identifications and confabulated freely. She was treated with injections of insulin and vitamin B_1, and the activity of the symptoms gradually moderated. However, a state of apparent confusion continued, characterized by a flippant and jocular attitude and the calling of people by wrong names. There was a "phoney" quality about this state which suggested that it was functional rather than organic. Therefore convulsant shock therapy was commenced in October, and after the first treatment a great improvement was noticed; this had continued, and at the time of the meeting the patient was well, although she still retained a flippant and jocular manner.

Dr. Evan Jones said that when this patient was admitted to hospital she had a Korsakow's psychosis, induced by alcoholic excess and probably aggravated by the excessive use of sedatives. She had slight signs of polyneuritis. The acute confusional stage of a Korsakow psychosis, which was similar to *delirium tremens*, usually subsided within a week or two if insulin was administered to stimulate the appetite and vitamin B_1 to counter the neuritic process. If the confusion continued in spite of such treatment, it might be due to one of two causes. The first was that the neuritic process might have passed the stage of reversibility, in which case signs of paresis or paralysis of the extremities would appear. The duration of such conditions ran into many months, pending the regeneration of demyelinized nerves. It was noteworthy that the administration of thiamin did not appear to have much effect in these cases. The second cause of continuance of the confusion was that the original toxic or organic confusion might have passed over into a functional psychosis; in that case shock therapy was indicated. The patient under consideration belonged in the second category.

NOTICE.

THE General Secretary of the Federal Council of the British Medical Association in Australia has announced that the following medical practitioners have been released from full-time duty with His Majesty's Forces and have resumed or will resume practice as from the dates mentioned:

Dr. A. Britten Jones, 175, North Terrace, Adelaide.
Dr. A. D. Reid, Mount Lofty, South Australia.
Dr. J. M. M. Pedler, 183, Payneham Road, St. Peters, South Australia.
Dr. A. M. Macintosh, 35, Gerrale Street, Cronulla, New South Wales (February 16, 1946).
Dr. M. W. Ginsberg, 351, New South Head Road, Double Bay (February 18, 1946).
Dr. J. M. Yeates, 135, Macquarie Street, Sydney (March 4, 1946).
Dr. N. R. Wyndham, 225, Macquarie Street, Sydney (February 1, 1946).
Dr. Athol Quayle, Wickham Terrace, Brisbane (January 2, 1946).
Dr. P. W. Hopkins, Gordon Street, Mackay, Queensland (February 18, 1946).
Dr. G. R. Kurrle, Samford Road, Alderley, Brisbane (January 2, 1946).
Dr. N. L. Hall, 285, Waterworks Road, Ashgrove, Brisbane (March, 1946).
Dr. Derick Yeates, 113, Wickham Terrace, Brisbane (February 11, 1946).
Dr. Oscar Alpins, 15, Collins Street, Melbourne (March 1, 1946).
Dr. W. L. Macdonald, 185, Macquarie Street, Sydney (February 1, 1946).

Correspondence.

THE LATE HERBERT MICHAEL MORAN.

SIR: Arrangements are being made for the publication of the many and interesting letters written by Dr. H. M. Moran in the course of his life. His father and sister have agreed to give their letters for publication, with the family matters eliminated of course. Dr. Scot Skirving has some, Dr. Fiaschi and I have a fair number. Between us we will link up the stories as told in the letters, and the book will possibly be called "The Life and Letters of Dr. H. M. Moran".

I would like to take this opportunity to ask anybody who has letters from Dr. Moran to let me have them. A rough draft will be made first and sent to the letter holders for their approval.

Any profits and royalties that may accrue are to be given to the University of Sydney to supplement a gift by Dr. Moran and his father for the endowment of lectures in the history of medicine. Nobody will receive any monetary gain by the publication of these letters.

Would you be kind enough to ask your readers to comply with this request of mine and let me have such correspondence as they may hold?

Yours, etc.,

E. H. MOLESWORTH.

Beanbah,
235, Macquarie Street,
Sydney.
February 6, 1946.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 28, of February 14, 1946.

AUSTRALIAN MILITARY FORCES.

Australian Army Medical Corps.

VX108488 Lieutenant-Colonel M. M. Rosefield is removed from the Regimental Supernumerary List and is appointed to command 111th Australian General Hospital, 19th November, 1945.

QX6264 Lieutenant-Colonel (Temporary Colonel) W. E. E. Langford relinquishes command of 2nd/14th Australian General Hospital and is placed upon the Regimental Supernumerary List, 3rd December, 1945.

VX63314 Captain (Temporary Major) I. A. N. McCallum relinquishes the rank of Temporary Major, 8th December, 1945.

The following officers are placed upon the Regimental Supernumerary List: QX6276 Lieutenant-Colonel D. McR. Yeates, 3rd December, 1945; NX217 Major (Temporary Lieutenant-Colonel) A. R. Colwell, 1st December, 1945; WX33952 Major W. P. White, 28th November, 1945, and NX70188 Captain E. M. Cortis, 11th November, 1945.

To be Temporary Colonel, 25th November, 1945.—NX183 Lieutenant-Colonel C. R. Blomfield.

2nd/2nd Australian General Hospital: To be Temporary Major, 28th November, 1945.—NX114068 Captain I. Monk.

2/9th Australian General Hospital.—NX76540 Major S. D. Allen is removed from the Regimental Supernumerary List, 29th November, 1945.

No. 113 (Concord) Military Hospital.—VX91068 Major T. E. Lowe is placed upon the Regimental Supernumerary List, 29th November, 1945.

Reserve of Officers.

The undermentioned officers are transferred to the Reserve of Officers with effect from the dates shown and where applicable cease to be seconded on the date prior to such transfer.

No. 113 (Concord) Military Hospital.—VFX141244 Captain D. M. Kiel, 17th October, 1945.

2nd/1st Australian Casualty Clearing Station.—VX20307 Major F. D. Stephens, D.S.O., 23rd October, 1945.

2nd Australian Field Ambulance (Australian Imperial Force).—VX145106 Captain M. Hain, 26th October, 1945.

NX71114 Captain L. O. S. Poideven, 31st October, 1945.

106th Australian General Hospital.—V145633 Captain W. R. Angus, 24th October, 1945.

Post-Graduate Work.

THE NEW SOUTH WALES POST-GRADUATE COMMITTEE IN MEDICINE.

COURSES IN SYDNEY.

THE following announcement is made by the New South Wales Post-Graduate Committee in Medicine concerning post-graduate courses.

Courses for General Practitioners.

A course for general practitioners is at present in progress and includes one week in obstetrics beginning on March 4 and ten days in pædiatrics beginning on March 11. A further course will commence on March 25 for ten weeks and will consist of the following: introductory, one week; special subjects, one week; medicine, two weeks; surgery, two weeks; gynæcology, one week; obstetrics, two weeks; and pædiatrics, one week. This course may be taken in whole or part, the fee being £2 12s. 6d. per week.

Course in Advanced Surgery.

A course in advanced surgery will be conducted until March 30 at a fee of £2 12s. 6d. per week. A further course will begin on July 29 for a duration of ten weeks and may be taken in whole or part.

Courses in Anatomy, Physiology and Biochemistry.

Courses in anatomy, physiology and biochemistry for the degree of Master of Surgery Part I and Parts I of the diplomas in ophthalmology, gynæcology and obstetrics, laryngology and oto-rhinology and psychological medicine are at present in progress and will be complete on March 15. Further courses for Parts I of the M.S., D.Oph., D.G.O. and D.L.O. will begin during Michaelmas Term.

Courses in Anæsthesia, Radiology, Advanced Medicine, Obstetrics and Gynæcology.

A course for the diploma in anæsthesia begins on March 18 for two terms. Fee: £25.

A course for the diploma in radiology (radiodiagnisis and radiotherapy) will begin on March 25 for three terms. Fee: £37 10s.

A course in advanced medicine (suitable for M.R.A.C.P. examination) will begin in July, an announcement concerning which will be made at a later date.

Courses in obstetrics and gynæcology of two weeks' duration will be conducted at the King George V Memorial Hospital from June 10 and September 9, 1946.

Attention is drawn to the fact that all the above courses are open to all medical practitioners, and it is requested that early application be made. Full information is obtainable from the Secretary of the Post-Graduate Committee, 131, Macquarie Street, Sydney. Telephone: BW 7483 or B 4606.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Cramer, John Earls, M.B., B.S., 1945 (Univ. Sydney), Tamworth Base Hospital, Tamworth.

Lee, Tom Herbert, M.B., B.S., 1942 (Univ. Sydney), 99, Spit Road, Mosman.

Obituary.

ALICE PRITCHARD CAMPBELL.

WE regret to announce the death of Dr. Alice Pritchard Campbell, which occurred on February 20, 1946, at Mount Victoria, New South Wales.

ROY LEPAGE MUECKE.

WE regret to announce the death of Dr. Roy LePage Muecke, which occurred on February 22, 1946, at Perth, Western Australia.

JOHN INGLIS CLARK COSH.

WE regret to announce the death of Dr. John Inglis Clark Cosh, which occurred on February 23, 1946, at Moss Vale, New South Wales.

THE FEDERAL MEDICAL WAR RELIEF FUND.

The following contributions to the Federal Medical War Relief Fund have been received.

New South Wales.

Sir C. Bickerton Blackburn, £250.
B. T. Edye, £105.
George W. Sinclair, £100.
Illawarra Suburbs Medical Practitioners' Emergency Service, £52 16s. 3d.
G. C. Willcocks, J. P. Hardie, £50.
S. R. Dawes, £26 5s.
F. T. Willard, W. F. Simmons, P. L. Hipsley, F. C. Florance, £25.
A. W. J. Stocks, V. M. Rich, A. O. Davy, B. P. Anderson-Stuart, £21.
Ruth and H. S. Stephens, A. T. Roberts, J. Macarthur, F. Barrington, £20.
Muriel B. McIlrath, Maurice Hurst, £15.
R. C. Winn, J. N. H. Wilson, T. H. Small, N. H. Rose, F. N. Rodda, H. J. Richards, H. R. G. Poate, R. J. G. A. Pittar, A. J. Ostinga, R. J. Nixon, R. A. Money, W. W. Martin, Maitland Medical Association, H. A. McCredie, Professor C. G. Lambie, W. L. Kirkwood, K. L. H. Kirkland, H. H. Johnston, R. J. Hunter, R. M. Glennie Holmes, R. I. Furber, W. M. A. Fletcher, B. H. Diamond, W. A. Conolly, J. N. Channon, A. W. J. Bulteau, C. G. Bayliss, G. M. Barron, Leo Bamber, C. J. B. Armstrong, J. B. Accola, £10 10s.
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Total: £2,025 15s. 3d.

Books Received.

"Hypnoanalysis", by Lewis R. Wolberg, M.D., with a Foreword by A. Kardiner, M.D.; 1945. New York: Grune and Stratton, Incorporated. 8¼" x 5¼", pp. 360. Price: $4.00.

"Biological Actions of Sex Hormones", by Harold Burrows, C.B.E., Ph.D., F.R.C.S.; 1945. Cambridge: Cambridge University Press. 9¼" x 6¼", pp. 534. Price: 42s. net.

"Essentials of Body Mechanics in Health and Disease", by Joel E. Goldthwait, M.D., F.A.C.S., LL.D., Lloyd T. Brown, M.D., F.A.C.S., Loring T. Swaim, M.D., and John G. Kuhns, M.D., F.A.C.S., with a chapter on the Heart and Circulation as Related to Body Mechanics by William J. Kerr, M.D., F.A.C.P.; Fourth Edition; 1945. Sydney: Angus and Robertson Limited. Philadelphia, London, Montreal: J. B. Lippincott Company. 7¾" x 5", pp. 351, with 128 illustrations. Price: 37s. 6d.

"Principles of Pediatrics and Pediatric Nursing", by Cecilia M. Knox, B.S., R.N.; 1945. Sydney: Angus and Robertson Limited. Philadelphia: F. A. Davis Company. 8½" x 5½", pp. 537, with illustrations, some of which are in colour. Price: 26s.

Diary for the Month.

March 2.—Tasmanian Branch, B.M.A.: Annual Meeting.
March 5.—Federal Council, B.M.A., in Australia: Meeting at Sydney.
March 5.—New South Wales Branch, B.M.A.: Organization and Science Committee.
March 6.—Western Australian Branch, B.M.A.: Council Meeting.
March 6.—Victorian Branch, B.M.A.: Branch Meeting.
March 7.—New South Wales Branch, B.M.A.: Special Groups Committee.
March 7.—South Australian Branch, B.M.A.: Council Meeting.
March 8.—Queensland Branch, B.M.A.: Council Meeting.
March 12.—Tasmanian Branch, B.M.A.: Ordinary Meeting.
March 12.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
March 19.—New South Wales Branch, B.M.A.: Medical Politics Committee.
March 19.—New South Wales Branch, B.M.A.: Ethics Committee.
March 20.—Western Australian Branch, B.M.A.: General Meeting.
March 21.—Victorian Branch, B.M.A.: Executive Meeting.
March 21.—South Australian Branch, B.M.A.: Council Meeting.
March 22.—Queensland Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

Medical practitioners are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All Public Health Department appointments.

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